AR TARGET SHEET

The following document was too large to scan as one unit, therefore, it has been broken down into sections.

EDMC#:	0025845
SECTION:	5 of 8
DOCUMENT #:	DOE/RL-92-67, DRAFT A
TITLE:	Final RI/FS Report for 1100-EM-1 OU, Hanford

3.0 HUMAN EXPOSURE ASSESSMENT

The purpose of the human exposure assessment is to estimate the magnitude, frequency, duration, and route of exposure to the COPC. The exposure estimation is used with appropriate toxicity information to assess the nature and extent of health threats from the COPC. The exposure assessment identifies receptor populations and exposure pathways as discussed in paragraph 3.1 through 3.4 below. This information is integrated with measured or estimated contaminant concentrations to quantify contaminant exposures and is presented in paragraph 3.5. A summary of the exposure assessment is provided in paragraph 3.7. For the BRSRA, a separate discussion of the evaluation of the exposures to lead at 1100-2 and HRL is discussed in paragraph 3.6.

3.1 IDENTIFICATION OF HUMAN RECEPTOR POPULATIONS FOR BISRA

Identification of the human populations at risk from exposure to COPC at the 1100-EM-1 subunits is usually determined by present and future land and water use assumptions. For the purposes of the BISRA, it is assumed that future land and water use will remain similar to existing conditions. The geographic distribution of the individual subunits throughout the operable unit limits the potential for the same receptor to have long-term exposures at multiple subunits. Currently, no workers are assigned to tasks in any of the subunits on a regular basis. However, the BISRA conservatively assumes that such assignments could occur in the future because the 1100-EM-1 Operable Unit is located in an area designated for industrial use and is surrounded by areas zoned, by the city of Richland, for industrial and commercial use.

Onsite industrial workers are selected as both the current and the future receptor populations for the 1100-EM-1 Operable Unit. Industrial workers are assumed to work full-time at only one subunit where they could potentially be exposed to contaminants from that subunit alone. The BISRA also assumes that personnel are assigned to the 1100 Area for purposes other than remediation. It is expected that the city of Richland's water will continue to be available to potential industrial facilities at the operable unit.

3.2 IDENTIFICATION OF HUMAN RECEPTOR POPULATIONS FOR THE BRSRA

There is no current residential use of 1100-EM-1, and none is expected in the future. However, as indicated previously, the EPA has requested evaluation of residential receptors at five subunits.

Onsite residents are evaluated as the receptor population for the 1100-EM-1 Operable Unit. Residents are assumed to live at only one subunit where they could potentially be exposed to contaminants from that subunit alone. It is also assumed that availability of city of Richland water at all subunits continues except at HRL. The hypothetical residents at HRL are conservatively assumed to use groundwater as the only source of potable water.

3.3 IDENTIFICATION OF EXPOSURE PATHWAYS FOR BISRA

The HSBRAM (DOE/RL-91-45) provides the exposure pathways that are used to evaluate the industrial scenario. The BISRA for the 1100-EM-1 Operable Unit is confined to the soil contamination. Potential exposure to groundwater contamination is not evaluated in the BISRA because potable water at 1100 Area facilities is currently obtained from the city of Richland. Although soil contaminants can leach to the groundwater and be transported to the Columbia River, workers in the 1100 Area would not use surface water directly from the Columbia River during the work day. Modeling presented in DOE/RL-90-18 also indicates that the concentrations of contaminants currently found in the groundwater in the vicinity of the 1100-EM-1 Operable Unit would undergo extensive dilution upon entering the Columbia River. Therefore, potential exposures to groundwater and surface water are not evaluated in the BISRA.

Although a few volatile organic compounds have been detected in the soil and/or soil gas, the evaluation of these contaminants in the Phase I RI Report indicates the inhalation of volatiles at the concentrations detected does not pose a risk greater than 1E-06 (DOE/RL-90-18). The EPA has also indicated that soil gas surveys are used for field screening and data generated from soil gas surveys should not be used in risk assessment (Einan, EPA [Letter to R. Stewart, DOE/RL] January 16, 1992, see appendix I). Given the above information, and because most volatile contaminants have been found only in soil gas at very low concentrations, the potential volatilization of contaminants from the soil is not considered an operable exposure pathway for the BISRA.

The potential exposure pathways through which industrial workers may be exposed to soil contaminants at a specific 1100-EM-1 subunit, are:

- Soil ingestion;
- inhalation of fugitive dust; and
- dermal exposure.

3.4 IDENTIFICATION OF EXPOSURE PATHWAYS FOR BRSRA

As defined by EPA [Einan, 1991 (see appendix I)] and a followup letter of clarification [Einan, 1992 (see appendix I], the exposure pathways for the BRSRA have been focused on contaminated soil. The soil-related pathways for BRSRA, specified by EPA, include the ingestion of soil, dermal contact with soil, ingestion of garden produce, and inhalation of particulates (*i.e.*, fugitive dust). Other potential soil-related pathways (*e.g.*, animal or crop uptake as associated with an agricultural scenario) were not requested by EPA [Einan, 1992 (see appendix I)].

Of the COPC specified by EPA for evaluation, three are classified as volatile contaminants that would generally be evaluated via the inhalation pathway. These are tetrachloroethane, trichloroethane, and 1,1,1-trichloroethane. For reasons outlined in paragraph 3.3, these contaminants will not be quanitatively evaluated in the BRSRA. The volatilization of contaminants from soil will be qualitatively addressed in paragraph 5.4.

EPA has directed that potential exposures through pathways associated with the groundwater at HRL be evaluated in the BRSRA [Einan, 1991 and Einan, 1992 (see appendix I)]. Pathways are evaluated for both direct groundwater use and for exposures through transport of contaminants off the Hanford Site. Currently, contaminants present in the groundwater in the vicinity of HRL may be transported to the Columbia River in the future. In the Phase I RI, the concentration of TCE at the groundwater interface with the river was estimated to be approximately 0.05 mg/L and at the city of Richland water intake, approximately 6E-06 mg/L. Additional modeling for the Phase II RI indicates TCE at the groundwater interface with the river would be less than 0.001 mg/L, which is less than the maximum contaminant level (MCL) for this contaminant.

In addition to the evaluation of direct groundwater use in the vicinity of HRL, EPA [Einan, 1992 (see appendix I)] directed that an evaluation of trichloroethane be conducted to assess the potential contribution to exposures for subunit residents who may swim in the Columbia River or eat fish from the Columbia River. Groundwater and surface water modeling results (DOE/RL-90-18), although shown to be conservative based on recent modeling done during the Phase II RI, are used to estimate potential concentrations of trichloroethane in surface water and fish. These conservative analyses result in an overestimation of potential risks associated with this pathway.

Columbia River water is used to recharge the North Richland well field to supplement potable water production. Groundwater modeling of contaminant transport to the Columbia River was presented in the Phase I RI Report. Evaluation of the risk associated with the ingestion of Columbia River water as a drinking source, as presented in the Baseline Risk Assessment in the Phase I RI report, indicates that the incremental lifetime cancer risk is approximately 2E-09 (DOE/RL-90-18). Therefore, further evaluation of the exposures through use of Columbia River water or city of Richland water is not presented for the BRSRA.

Given the above information, the soil-related exposure pathways evaluated in the BRSRA for onsite residential receptors at the five specified 1100-EM-1 subunits under consideration are:

- Soil ingestion;
- inhalation of fugitive dust;
- ingestion of garden produce; and
- dermal exposure to soil.

In addition, EPA specified groundwater exposure pathways for contaminants detected in groundwater in the vicinity of HRL include:

- Ingestion of groundwater;
- inhalation of volatiles from groundwater;
- ingestion of Columbia River fish; and
- dermal contact with Columbia River water during swimming.

The dermal route of exposure to potable water was also considered, but is not included in the exposure pathway because the dermal route has been reported as insignificant, tetrachloroethane (EPA 1985), and 1,1,1-trichloroethane (ATSDR 1988). It is not clear whether exposure to dermal nitrate is a concern. This issue is discussed in the uncertainty analysis paragraph 5.4.

3.5 QUANTIFICATION OF EXPOSURES FOR BISRA AND BRSRA

The exposure assessment includes a quantification of exposures for the receptors via the exposure pathways that have been identified. An exposure concentration (*i.e.*, a concentration that is contacted over the exposure period) is estimated and used with population variables (*e.g.*, exposure parameters) and assessment variables (*e.g.*, averaging times) to determine an intake. The following paragraphs describe the assumptions, information, and calculations used to estimate exposure intakes for onsite residents and industrial workers. A detailed presentation of sample calculations is provided in appendix IV.

3.5.1 Exposure Concentrations

3.5.1.1 Soil Ingestion and Dermal Exposure Pathways for BRSRA and BISRA--The exposure concentrations for the soil ingestion and dermal exposure pathways are conservatively assumed to be the maximum concentrations of the COPC as determined from the Phase I RI Report or Phase II soil sampling (see tables 2-3 through 2-9). The use of the maximum concentration is generally conservative because it does not consider any actual or potential spatial distribution of the contaminant over the subunit (*i.e.*, it is highly likely that the concentration at the actual exposure location will be significantly less than the maximum). This assumption is also conservative because it assumes that the maximum

concentration is readily accessible for receptor contact even if the maximum concentration is actually located below the surface at considerable depth.

3.5.1.2 Inhalation - Fugitive Dust for BISRA and BRSRA--Exposure concentrations for the fugitive dust pathway are derived using subunit specific maximum soil concentrations and the subunit specific fugitive dust concentration in air at the receptor location. Exposure concentrations in air are not determined for the 1100-4 subunit because the contaminated soil is located beneath a cement floor, inside the 1171 Building.

The fugitive dust concentration is calculated by incorporating a subunit specific emission rate and deposition rate into EPA's FDM (version 91109 and Bowman Environmental Engineering, version 1.21). The FDM uses site-specific meteorological data and has the capability to directly compute the effect of wind speed on each source-specific emission rate during each meteorological averaging period. The site-specific meteorological data used by the FDM consists of 1 full year (1988) of hourly wind speed and direction data, (collected at a monitoring station located near the 1100-EM-1 operable unit), and hourly temperature, mixing height, and stability class data, (measured at the Hanford Meteorological Station). The FDM also accounts for deposition of suspended particulates during airborne travel.

Fugitive dust emission rates are calculated for each source using the Universal Soil Loss equation as simplified by Woodruff and Siddoway, 1965:

$$EF = AIKCL'V'$$

where EF is the emission factor (tons/acre-yr), A is that portion of total dust emissions that would be measured as suspended particulate matter and is typically defined as particles with a diameter less than 30 microns. The value of A is 0.041 (dimensionless), for fine soils (Baskett, 1983). I is the soil erodibility factor (tons/acre-yr), K is the surface roughness factor (dimensionless), C is a climatic factor (dimensionless), L' is the unsheltered field width factor (dimensionless), and V' is the vegetative cover factor (dimensionless).

The soil erodibility factor, I, is determined from table 3 of *Wind Erosion Forces In The United States and Their Use in Predicting Soil Loss* (Skidmore and Woodruff, 1968) and is based on the portion of surface soil retained by a No. 20 standard sieve with 0.84 mm (0.03 inches) square mesh. Conservatively biased estimates of the >0.84 mm (0.03 inches) fractions of the surface soils for the 1100-2, 1100-3, UN-1100-6 subunits, and HRL subunits are 35, 37, 5, and 29 percent, respectively. These data are obtained from paragraph 3.5.2.2 of the Phase I RI Report (DOE/RL-90-18), and convert to I values of 65, 62, 180, and 76 tons/acre-yr. For the Ephemeral Pool, grain size distribution data are not available. The fraction >0.84 mm (0.03 inches) at this subunit is conservatively assumed to be 5 percent (the same as UN-1100-6 subunit), resulting in an I value of 180 tons/acre-yr.

The surface roughness factor, K, accounts for the resistance to wind erosion provided by ridges and furrows and is conservatively assumed to be unity (*i.e.*, no reduction in resistance).

An unsheltered field width factor, L, of .7 is typical for exposed areas about 305 m (1,000 feet) across (Baskett, 1983).

A vegetative cover factor of unity is conservatively assumed, making no allowance for reductions in emissions due to vegetation.

Wind velocity and soil moisture contribute significantly to windblown fugitive dust emission rates and relate to the climatic factor, C, as:

$$C = 0.345u^3/PE^2$$

where u^3 is the wind velocity in miles per hour and PE^2 is the site-specific Thornthwaite's precipitation-evaporation index. A PE^2 value of 29.1 was assumed (U.S. Weather Bureau and SCS 1962). Meteorological records were used to determine u^3.

Because the climatic factor varies as the cube of the wind velocity, windspeed greatly affects the emission rate. However, windspeed varies significantly with time. The FDM model has the capability to directly compute the effect of windspeed on the emission rate for each source during each meteorological averaging period. Therefore, the emission rate is entered into the model as a conservative source-specific coefficient with wind speed as the only variable:

$$EF = (0.041)I(0.7)(0.345)u^3/(29.1)^2$$

The entire suspended particulate fraction is conservatively regarded as respirable. These emission rates are used in the FDM to determine downwind air concentrations of respirable fugitive dust.

In order to estimate the concentration of fugitive dust at a receptor location, the FDM accounts for gravitational settling and particle disposition during airborne travel. A "default" particle size distribution in the FDM test input data, listed below, was used.

Particle Size Class	Particle Diameter (μm)	Fraction in Each Size Class
1	1.25	0.0262
2	3.75	0.0678
3	7.50	0.1704
4	12.50	0.1536
5	20.00	0.5820

A particle density of 2.5 g/cm^3 was used, which is consistent with the range for most mineral soils (Brady, 1984). Comparison of dust concentrations calculated by the FDM, with particle densities ranging from 0 to 2.5 g/cm^3, showed very little sensitivity to this parameter; dust concentrations typically varied only hundredths of a μg/kg between high and low particle densities.

The receptor location within each subunit was chosen as the point calculated by the FDM with the maximum fugitive dust concentration, based on a 25 meter grid system (50 m

for HRL) that was centered on the middle of the subunit. The concentration of fugitive dust at each subunit as calculated by the FDM, is shown in table 3-1. The contaminant concentration in air is assumed to be directly proportional to the contaminant concentration in soil. Therefore, the fugitive dust concentrations were multiplied by the maximum soil contaminant concentrations (table 2-1), and appropriate conversion factors, to produce contaminant concentrations in air shown in table 3-2.

3.5.1.3 Garden Produce Pathway for BRSRA--The quantification of exposures from the ingestion of garden produce requires an estimation of the contaminant concentration in the produce. The amount of contaminant that is taken up by garden produce from the soil can be estimated using published or derived plant uptake factors for specific contaminants. The *Land Application and Distribution and Marketing of Sewage Sludge, Technical Support Document* (EPA, 1986a) provides plant-specific contaminant uptake factors for a number of contaminants. Uptake factors are available for both inorganic and organic contaminants for a variety of garden or agricultural plants. The relationship between the contaminant concentration in soil to a contaminant concentration in plants is:

$$\text{Plant concentration} = \text{Uptake factor} \times \text{soil concentration}$$

where the uptake factor is expressed as [μg/g tissue dry weight (μg/g soil)$^{-1}$] and the soil concentration is expressed as μg/g. All soil concentrations are expressed as dry weight. The evaluation takes into account only the contamination present in soil and does not include any potential contributions from irrigation water.

Four specific garden produce categories are evaluated in this BRSRA based on the direction provided by EPA [Einan, 1991; Einan, 1992 (see appendix I)]. The garden produce categories and corresponding vegetables evaluated are: root (*e.g.*, carrots), potatoes, leafy vegetables (*e.g.*, lettuce), and garden fruits (*e.g.*, tomatoes).

A summary of the plant uptake factors for the COPC is provided in table 3-3. Contaminant-specific uptake factors are available for arsenic, PCB's, and lead. The following assumptions were made in the absence of more appropriate data. The plant uptake factors for BEHP are conservatively assumed to be the same as for PCB's. The plant uptake factors for heptachlor are used as a conservative surrogate for chlordane. Chlordane is chemically similar to heptachlor and contains approximately 10 percent heptachlor (by weight). However, the root uptake factor for chlordane is conservatively derived as a 95 percent UCL for a variety of uptake factors for chlordane in sugar beets (EPA, 1986a).

Since uptake factors for chromium and beryllium are not reported in EPA, (1986a), uptake factors for arsenic, cadmium, lead, mercury, nickel, selenium, and zinc are used to derive a conservative estimate [*i.e.*, upper 95 percent confidence limit (UCL)] for each specific plant category. These values are used as a surrogate plant uptake factor for chromium and beryllium, except the uptake factor for chromium in leafy vegetables, which is published in Kabata-Pendias and Pendias, 1984.

Table 3-1. Fugitive Dust Concentration for Specific 1100-EM-1 Operable Subunits[1].

Subunit	Fugitive Dust Concentration ($\mu g/m^3$)
1100-1	0.0032
1100-2	3.17
1100-3	2.37
UN-1100-6	1.58
Horn Rapids Landfill	9.93
Ephemeral Pool	4.23

[1] Based on the maximum concentration generated by the EPA Fugitive Dust model.

Table 3-2. Estimated Air Concentrations Based on Maximum Contaminant Concentrations.

Contaminant	1100-1	1100-2	1100-3	UN-1100-6	Horn Rapids Landfill	Ephemeral Pool
	Downwind Air Concentration (mg/m^3)	Downwind Air Concentration (mg/m^3)	Downwind Air Concentration (mg/m^3)	Downwind Air Concentration (mg/m^3)	Downwind Air Concentration (mg/m^3)	Downwind Air Concentration (mg/m^3)
Antimony	--	--	--	--	--[a]	--
Arsenic	1.0E-11	--	8.1E-09	--	6.6E-08	--
Barium	--	--	--	--	1.3E-05	--
Beryllium	--	--	--	--	1.1E-08	--
Chromium	--	5.3E-08	3.3E-08	--	1.2E-05	--
Copper	--	--	--	--	--[a]	--
Lead	--	--	6.3E-08	--	--	--
Nickel	--	--	--	--	5.5E-06	--
Thallium	--	--	--	--	--[a]	--
Vanadium	--[a]	--	--	--	--[a]	--
Zinc	--	--	--	--	--[a]	--
BEHP	--	--	--	4.0E-05	--	--
Beta-HCH	--	--	--	--	9.3E-10	--
Chlordane	--	--	--	2.9E-09	--	1.1E-08
DDT	--	--	--	--	2.0E-08	--
Heptachlor	--	--	--	1.0E-10	2.0E-10	1.2E-10
PCBs	--	--	--	--	1.0E-06	1.8E-07
Tetrachloroethane		1.1E-10			6.0E-11	

[a]Inhalation RfDs and SFs are not available with which to evaluate these contaminants of potential concern.
-- Indicates not a contaminant of potential concern for the air pathway at this subunit.

The potential exposure to volatile compounds through the garden scenario was not quantitatively evaluated in the BRSRA and volatile contaminants are not presented in table 3-3. Volatile compounds such as tetrachloroethane have short half-lives in soil, are not persistent in the soil, and are not expected to be readily taken up by plants or to bioaccumulate in plants (Ryan *et al.*, 1988). Based on the very low concentrations of volatile contaminants detected in the soil, plant uptake of these COPC is not considered an operable exposure route and the uptake of volatile contaminants from soil or soil gas is not evaluated further in this BRSRA.

A summary of contaminant concentrations for the garden pathway is provided in table 3-4. The 1100-2 subunit is not presented in this table because the only contaminant at this subunit (tetrachloroethane) is not evaluated for the garden pathway.

3.5.1.4 <u>Groundwater Pathways for BRSRA</u>--The exposure concentrations for the groundwater ingestion and inhalation of volatiles from groundwater pathways are the maximum concentrations of the COPC as determined from the Phase I and Phase II groundwater sampling at HRL (DOE/RL-90-18 and appendix 5). The concentrations are 0.11 mg/L and 61 mg/L for trichloroethane and nitrate as N, respectively. As directed, 1,1,1-trichloroethane was not evaluated [Einan, 1991 (see appendix I)].

3.5.4.5 <u>Residential-Related Recreational Pathways for BRSRA</u>--Two recreational exposures are evaluated for residents at HRL. Residents may swim in or consume fish from the Columbia River, which could be potentially impacted by groundwater from the vicinity of the 1100-EM-1 Operable Unit. Trichloroethane and nitrate are groundwater COPC that potentially could be transported to the Columbia River. Only trichloroethane is likely to be dermally absorbed. The estimated future concentration of trichloroethane in the Columbia River is conservatively assumed to be 6E-06 mg/L (DOE/RL-90-18), which is the value used in evaluating potential dermal exposures during swimming. By extrapolating observations of groundwater concentrations, the nitrate value at the Columbia River is estimated to be 0.003 mg/L. This is below the MCL of 10 mg/L for nitrate.

As trichloroethane may bioaccumulate in fish, with a bioconcentration factor of 17 L/kg on a wet weight basis (EPA, 1986b), the resulting contaminant concentration in the tissue of fish inhabiting the Columbia River in the vicinity of the city of Richland water intake is conservatively estimated to be 1E-04 mg/kg. Little information exists on the bioconcentration potential of nitrate in animals. However, because nitrate is readily metabolized, bioconcentration is unlikely. Therefore, nitrate is not evaluated for exposures through bioaccumulation in fish.

Table 3-3. Summary of Plant Uptake Factors[a,b].

Contaminant	Leafy	Root	Garden Fruits	Potatoes
Arsenic	0.04	0.02	0.002	0.0006
BEHP[c]	0.38	0.36	0.02	0.02
Beryllium[d]	0.43	0.26	0.041	0.06
Chlordane	0.02[e]	2.02[f]	0.21[e]	0.3[e]
Chromium	0.2[g]	0.26[d]	0.041[d]	0.06[d]
Lead	0.008	0.003	0.002	0.0008
PCBs	0.38	0.36	0.02	0.02

[a]All uptake factors expressed as [μg/g tissue Dry Weight (μg/g soil)$^{-1}$]
[b]Source: EPA 1986a unless otherwise indicated
[c]PCB uptake factors used as surrogates for BEHP
[d]95% upper confidence limit of mean for uptake factors of As, Cd, Pb, Hg, Ni, Se, Zn (EPA 1986a)
[e]Heptachlor uptake factors used as surrogates for chlordane
[f]95% upper confidence limit of mean for uptake of chlordane by sugar beets
[g] Kabata - Pendias and Pendias 1984

Table 3-4. Summary of Contaminant Concentrations for the Garden Pathway at Specific 1100-EM-1 Operable Subunits Based on Maximum Contaminant Concentrations.

	Leafy (lettuce) (mg/kg)	Root (carrots) (mg/kg)	Garden Fruits (tomatoes) (mg/kg)	Potatoes (mg/kg)
1100-3				
Arsenic	1.36E-01	6.8E-02	6.8E-03	2.0E-03
Chromium	2.8E+00	3.6E+00	5.7E-01	8.4E-01
Lead	2.11E-01	7.9E-02	5.3E-03	2.1E-02
UN-1100-6				
BEHP	9.5E+03	9.0E+03	5.0E+02	5.0E+02
Chlordane	3.7E-02	3.8E+00	3.9E-01	5.6E-01
Ephemeral Pool				
Chlordane	5.6E-02	5.7E+00	5.9E-01	8.4E-01
PCB	1.6E+01	1.5E+01	8.4E-01	8.4E-01
Horn Rapids Landfill				
Arsenic	2.6E-01	1.3E-01	1.3E-02	4.0E-03
Beryllium	5.6E-01	3.4E-01	5.3E-02	7.8E-02
Chromium	2.5E+02	3.2E+02	5.1E+01	7.5E+01
Lead	6.8E+00	2.6E+00	1.7E+00	6.8E-01
PCB	3.9E+01	3.7E+01	2.0E+00	2.0E+00

3.5.2 Calculation of Contaminant Intakes

Standard EPA equations for calculation of intakes, as provided in RAGS (EPA, 1989a) and the HSBRAM (DOE/RL-91-45 1991) are used as the basis for all intake calculations. The table 3-4 basic equation for calculating intakes, normalized with respect to body weight, for ingestion or inhalation is:
where:

$$\text{Intake} = \frac{C \times IR \times EF \times ED \times CF}{BW \times AT}$$

Intake	=	chronic daily intake of the contaminant (mg/kg-d)
C	=	concentration of contaminant in the medium (*e.g.*, mg/kg or mg/m^3)
IR	=	intake rate (*e.g.*, mg/d or m^3/d)
EF	=	exposure frequency (d/yr)
ED	=	exposure duration (yr)
BW	=	body weight (kg)
AT	=	averaging time (d/yr x yr)
CF	=	conversion factor (as appropriate)

The exposure parameters (*i.e.* body weight, averaging time, contact rate, exposure frequency, and exposure duration) for the BISRA are those presented for the industrial scenario of the HSBRAM (DOE/RL-91-45), and are discussed below with conversion factors indicated, as appropriate. A summary of the industrial and residential exposure parameters are provided in table 3-5 and table 3-6, respectively. See appendix IV for specific BRSRA calculations, *e.g.*, in combining child and adult exposures.

3.5.2.1 Soil Ingestion for BISRA--

C	=	maximum contaminant concentration (mg/kg)
IR	=	intake rate (50 mg/d)
EF	=	exposure frequency (146 d/yr)
ED	=	exposure duration (20 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 20 yr; carcinogenic effects: 365 d/yr x 70 yr)
CF	=	conversion factor (1E-06 kg/mg)

3.5.2.2 Soil Ingestion for BRSRA--All exposure parameters for the soil ingestion pathway are those presented for the residential scenario as discussed in *Supplemental Risk Assessment Guidance for Superfund*, (EPA-10, 1991). These factors are used as required by EPA [Einan, 1992 (see appendix I)]. For evaluating both carcinogens and noncarcinogens, the exposure assumptions are based on a child and an adult exposure.

C	=	maximum contaminant concentration (mg/kg)
IR	=	intake rate (Child: 200 mg/d; Adult: 100 mg/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (Child: 6 yr; Adult: 24 yr)
BW	=	body weight (Child: 15 kg; Adult: 70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)
CF	=	conversion factor (1E-06 kg/mg)

3.5.2.2 Inhalation of Fugitive Dust for BISRA--The following are exposure parameters used for the industrial scenario:

C	=	estimated air concentration (mg/m^3)
IR	=	intake rate (20 m^3/d)
EF	=	exposure frequency (250 d/yr)
ED	=	exposure duration (20 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 20 yr; carcinogenic effects: 365 d/yr x 70 yr)

3.5.2.3 Inhalation of Fugitive Dust for BRSRA--All exposure parameters for the inhalation of fugitive dust pathway are those presented for the residential scenario, as discussed in EPA-Region 10 guidance (EPA-10 1991). For evaluating both carcinogens and noncarcinogens, the exposure assumptions are based on an adult exposure.

C	=	estimated air concentration (mg/m^3)
IR	=	intake rate (20 m^3/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)

3.5.2.4 Dermal Exposure to Contaminated Soil for BISRA--The intake equation provided in paragraph 3.5.2 above, is modified to provide the absorbed dose equation for dermal exposures to contaminated soil. For the purpose of the BISRA, it is conservatively assumed that workers do not wear protective clothing that would limit dermal exposure. Exposure factors, as provided in the HSBRAM (DOE/RL-91-45 1992), are indicated.

Table 3-5. Summary of Industrial Scenario Exposure Factors.

Exposure Factor	HSBRAM Reasonable Maximum Exposure[a]
Intake Rate	
Soil Ingestion	50 mg/d
Inhalation	20 m^3/d
Exposure Frequency	
Soil Ingestion	146 d/yr
Inhalation	250 d/yr
Dermal	146 d/yr
Exposure Duration	20 yr
Body Weight	70 kg
Averaging Time	
Carcinogens	70 yr x 365 d/yr
Noncarcinogens	20 yr x 365 d/yr
Skin Surface Area	5000 cm^2
Soil-to-Skin Adherence Factor	0.2 mg/cm^2/event
Absorption Factor	
Inorganics	0.001[c]
BEHP	0.0055[b]
All other organics	0.06[c]

[a]DOE-RL 1992; factors based on EPA-10 (1991), WAC 173-340, EPA (1992c)
[b]Calculated; see Subsection 3.5.3
[c]EPA (1992c)

Table 3-6. Summary of Residential Scenario Exposure Factors.

Exposure Factor	Reasonable Maximum Exposure[a]
Intake Rate	
Ingestion	
Adult - Soil	100 mg/d
Child - Soil	200 mg/d
Adult - Groundwater	2 L/d
Inhalation	
Adult - Soil	20 m^3/d
Adult - Groundwater (volatiles)	15 m^3/d
Fish Ingestion[c]	54 g/d
Garden Produce[b]	
Root (e.g., carrots)	0.88 g/d
Leafy (e.g., lettuce)	1.1 g/d
Garden fruit (e.g., tomato)	2.2 g/d
Potato	9.1 g/d
Exposure Frequency	350 d/yr 2.6 hr/d, 7 d/yr (swimming)
Exposure Duration	
Soil Ingestion and Dermal	
Adult	24 yr
Child	6 yr
All other pathways	30 yr
Body Weight	
Adult	70 kg
Child	15 kg
Averaging Time	
Carcinogens	70 yr x 365 d/yr
Non-carcinogens	30 yr x 365 d/yr
Skin Surface Area	
Adult - Soil	5000 cm^2 (summer); 1900 cm^2 (winter)
Child - Soil	3900 cm^2
Adult - Swimming	20,000 cm^2
Soil to Skin Adherence Factor	1 mg/cm^2/d
Contaminant-Specific Absorption Factor	
Inorganics[d]	0.001
BEHP[e]	0.0055
All other organics[d]	0.06
Permeability Coefficient - Trichloroethene[d]	4E-01 cm/hr
Groundwater Volatilization Factor[e]	0.5 L/m^3

[a]Factors based on EPA-10 (1991) unless otherwise specified
[b]EPA (1986a)
[c]EPA (1991a)
[d]EPA (1992c)
[e]Calculated factor; see Section 3.5.3

$$\text{Dermally absorbed dose} = \frac{CS \times SA \times AF \times ABS \times EF \times ED \times CF}{BW \times AT}$$

where:

Dermally absorbed dose = (mg/kg-d)

CS	=	maximum concentration of contaminant in soil (mg/kg)
SA	=	skin surface area available for contact (5000 cm^2)
AF	=	soil-to-skin adherence factor (0.2 mg/cm^2/event)
ABS	=	contaminant-specific absorption factor (unitless)
EF	=	event frequency (146 events/yr)
ED	=	exposure duration (20 yr)
CF	=	conversion factor (1E-06 kg/mg)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 20 yr; carcinogenic effects: 365 d/yr x 70 yr)

The contaminant-specific absorption factor is a value that is either assumed or derived from published literature. Many factors influence the dermal absorption of contaminants from the soil. Some of these factors include the amount of soil adsorbed to the skin, the contact time of the soil with the skin (time between exposure and washing), chemical properties of the contaminants, and the condition of the skin. Contaminants bound to a soil matrix are less bioavailable than pure or dilute solutions of contaminants applied directly to skin. Specific information on dermal absorption for most of the COPC is limited.

For the inorganic COPC, a review of the published literature, including available toxicological profiles from the Agency for Toxic Substances and Disease Registry (ATSDR), indicates that uptake across intact skin is very limited for most metal ions. The average dermal absorption of cadmium from a soil matrix is estimated at 1.0 percent (*i.e.*, 0.01), as discussed in the *"Dermal Exposure Assessment: Principles and Applications"* (EPA, 1992c). Therefore, for the calculation of dermal intakes, it is assumed that the contaminant-specific absorption factor (ABS) is 0.01 for all inorganic COPC based on the available information for cadmium.

Several organic COPC are also present in the soil at UN-1100-6 subunit, HRL, and the Ephemeral Pool. A review of the literature provided little specific information on the absorption of the specific organic compounds of potential concern — beta-HCH, chlordane, DDT, heptachlor, and PCB's — from skin contact with contaminated soil. EPA (1992c) recommends the use of an upper bound estimate of 6 percent (*i.e.*, 0.06), as an absorption factor for PCB's based on studies of 3,3', 4,4' tetrachlorobiphenyl. A value of 0.06 is assumed to be an appropriate ABS for all organic COPC except BEHP.

For BEHP, data are available on potential dermal absorption that can be combined with assumptions of contact time with the soil and dermal bioavailability. Studies in rats have shown that 6.9 percent of BEHP, applied as pure product, is dermally absorbed (Life Systems, Inc. 1989). Ryan *et al.* (1987) suggest that only 10 percent of organic

contaminants in soil are generally bioavailable for dermal exposures. If the contact time with the soil is conservatively assumed to be 8 hr, 80 percent of the contaminant is estimated to be absorbed during this time period (Shu *et al.* 1988). This absorption information and dermal bioavailability information can be combined to estimate an absorption factor for BEHP of 0.55 percent (*i.e.*, 0.0055).

3.5.2.5 Dermal Exposure to Contaminated Soil for BRSRA--The equation and assumptions used for the BRSRA for dermally absorbed dose is the same as that used for the BISRA, as described above. The dermal exposure parameters for the contaminated soil pathway are those recommended by EPA (EPA-10, 1991). For evaluating both carcinogens and noncarcinogens, the exposure assumptions are based on a child and an adult exposure. The following parameters are different for the BRSRA:

SA	=	skin surface area available for contact (Child: 3900 cm^2, Adult: 5000 cm^2 - summer, 1900 cm^2 - winter)
AF	=	soil-to-skin adherence factor (1 mg/cm^2/event)
EF	=	event frequency (Child: 1/event/day, 350 d/yr; Adult: 1/event/day, 350 d/yr with 90 d as summer and 260 d as winter)
ED	=	exposure duration (Child: 6 yr; Adult: 24 yr)
BW	=	body weight (Child: 15 kg; Adult: 70 kg)

3.5.2.6 Ingestion of Garden Produce for BRSRA--The exposure parameters for the ingestion of garden produce [*i.e.*, dry weight (DW) consumption rates for each vegetable category) have been derived from EPA (1986a, 1989d, and 1990]. The percentage of homegrown vegetables and the remaining exposure factors are provided in EPA (1991a). The currently recommended parameters in EPA (1991a) are based on fresh weight and are for total vegetable consumption only. Therefore, they are not appropriate to evaluate individual vegetable categories or for use with plant uptake factors based on DW.

For evaluating both carcinogens and noncarcinogens, the exposure assumption is based on an adult exposure. The highest daily DW consumptions for a vegetable category provided in EPA (1986a, 1989d, and 1990) have been adjusted to represent the amount that is homegrown (*i.e.*, 40 percent), as recommended in EPA (1991a). The adjusted daily DW consumptions are:

- Root (*e.g.*, carrots) 0.88 g/d
- Leafy (*e.g.*, lettuce) 1.1 g/d
- Garden fruits (*e.g.*, tomatoes) 2.2 g/d
- Potatoes 9.1 g/d

The exposure parameters for the garden produce pathway are:

C	=	estimated plant concentration (mg/kg dry weight)
IR	=	intake rate (produce-specific g/d dry weight)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)
CF	=	conversion factor (1E-03 kg/g)

3.5.2.7 Ingestion of Groundwater for BRSRA--The exposure parameters for the consumption of groundwater are those presented for the residential scenario, as discussed in EPA-Region 10 guidance (EPA-10, 1991). For evaluating both carcinogen and non-carcinogens, the exposure assumptions are based on an adult exposure.

CW	=	estimated concentration in groundwater (mg/L)
IR	=	intake rate (2 L/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)

3.5.2.8 Inhalation of Volatiles from Groundwater for BRSRA--The potential inhalation of volatiles from the use of groundwater in a residence is evaluated as recommended in EPA-Region 10 guidance (EPA-10, 1991). Exposure assumptions are based on an adult exposure:

CW	=	estimated concentration in water (mg/L) x K volatilization factor (0.5 L/m^3)
IR	=	intake rate (15 m^3/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)

3.5.2.9 Fish Ingestion for BRSRA--The exposure parameters for the consumption of fish are those presented in EPA (1991a). Exposure assumptions are based on adult exposure:

C	=	estimated concentration in fish (mg/kg)
IR	=	intake rate (54 g/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)
CF	=	conversion factor (1E-03 kg/g)

3.5.2.10 Dermal Contact with Columbia River Water for BRSRA--The intake equation provided above for ingestion is modified to provide the absorbed dose equation for dermal contact with Columbia River water. All exposure parameters for the dermal contact with water are those presented for the residential scenario as discussed in EPA-Region 10 guidance (EPA-10, 1991). For evaluating both carcinogens and noncarcinogens, the exposure assumptions are based on an adult exposure.

$$\text{Dermally absorbed dose} = \frac{CW \times SA \times K_p \times ET \times EF \times ED \times CF}{BW \times AT}$$

where :

Dermally absorbed dose = normalized with respect to bodyweight (mg/kg-d)

CW	=	maximum concentration of contaminant in water (mg/L)
SA	=	skin surface area available for contact (20,000 cm^2)
K_p	=	contaminant-specific permeability coefficient (cm/hr)
ET	=	event time (2.6 hr/d)
EF	=	event frequency (7 d/yr)
ED	=	exposure duration (30 yr)
CF	=	conversion factor (1 L/1000 cm^3)
BW	=	body weight (70 kg)
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)

The contaminant-specific permeability factor (K_p) is a value that is either assumed or can be derived from the literature if sufficient dermal absorption information is available. Trichloroethane is the only contaminant of potential concern that may impact the Columbia River that is evaluated for the BRSRA. The K_p for trichloroethane is 2E-01 cm/hr (0.08 in/hr) (EPA, 1992c).

3.5.3 Summary of Contaminant Intakes for BISRA

The estimated intakes of COPC for industrial workers are provided in tables 3-7 and 3-8. The intakes are provided for both noncarcinogenic effects and carcinogenic effects. Specific intakes are not presented if there is no RfD or SF for a contaminant, or if the SF is zero (*e.g.*, a contaminant is not carcinogenic by this pathway). It should be noted that the fugitive dust intake of arsenic is reduced by 30 percent because of absorption (EPA, 1992b).

3.5.4 Summary of Contaminant Intakes for BRSRA

The estimated intakes of COPC for onsite residents are provided in tables 3-9 through 3-12. As noted for the BISRA, specific intakes are not presented if there is no risk SF or RfD for a contaminant or if the slope factor is zero. Table 3-9 provides the intakes for the soil ingestion, inhalation of fugitive dust, and dermal contact pathways based on the maximum contaminant concentrations. As referenced for the BISRA, fugitive dust intake of arsenic is reduced by 30 percent because of absorption. The contaminant intakes from the consumption of garden produce are presented in table 3-10. The 1100-2 subunit is not presented in table 3-10 because the only contaminant at this subunit (tetrachloroethane) is not evaluated for the garden pathway. Volatile contaminants detected at HRL are not presented in this table. Contaminant intakes for the groundwater pathway are shown in table 3-11. The contaminant intakes from the residential-related recreational pathways (consumption of fish and dermal absorption through swimming) are presented in table 3-12. The intakes are provided for both noncarcinogenic effects and carcinogenic effects.

3.6 EXPOSURE ASSESSMENT FOR LEAD IN BRSRA

EPA (see chapter 2 and appendix I) has indicated that lead is to be evaluated as a contaminant of potential concern at 1100-3 and at HRL, where it is detected in soil at a maximum concentration of 26.4 mg/kg, and 854 mg/kg, respectively. The EPA has also recommended the use of the Uptake/Biokinetic Model (UBK) for evaluating the potential residential exposures to lead at these two subunits.

EPA does not currently recommend numerical toxicity values for lead, in part, because there is no scientific consensus concerning the effects of lead at low doses. Data on blood-lead levels and various health effects indicate a spectrum of adverse health effects in populations having increased blood-lead levels. EPA has reviewed key studies relating to the toxicokinetics and health effects of lead in humans in its *Air Quality Criteria Document for Lead* (EPA, 1986c). Although a threshold for these effects has not been established, the available evidence suggests that it lies within 10 to 15 μg/dl. The evidence for adverse effects below this range of blood-lead is uncertain and remains controversial.

Table 3-7. Summary of Industrial Scenario Intakes Based on Maximum Contaminant Concentrations for 1100-1, 1100-2, 1100-3, 1100-4, UN-1100-6, and the Ephemeral Pool.

Contaminant	Pathway					
	Soil Ingestion (mg/kg-d)		Fugitive Dust Inhalation (mg/kg-d)		Dermal Exposure (mg/kg-d)	
	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic
1100-1						
Arsenic	9.3E-07	2.6E-07	--	1.7E-13*	1.8E-08	5.2E-09
Vanadium	3.4E-05	--	--	--	6.7E-07	--
1100-2						
Chromium	4.8E-06	--	--	3.0E-09	9.5E-08	--
1100-3						
Chromium	4.0E-06	--	--	1.9E-09	7.9E-08	--
1100-4						
Arsenic	1.7E-06	4.7E-07	--	--	3.3E-08	9.4E-09
Beryllium	2.7E-07	7.6E-08	--	--	5.3E-09	1.5E-09
UN-1100-6						
BEHP	7.2E-03	2.0E-03	--	2.2E-06	7.9E-04	2.2E-04
Chlordane	5.3E-07	1.5E-07	--	1.6E-10	6.4E-07	1.8E-07
Heptachlor	1.9E-08	5.3E-09	--	5.8E-12	2.2E-08	6.4E-09
Ephemeral Pool						
Chlordane	8.1E-07	2.3E-07	--	6.6E-10	9.6E-07	2.7E-07
Heptachlor	8.4E-09	2.4E-09	--	6.9E-12	9.9E-09	2.8E-09
PCBs	--	3.4E-06	--	1.0E-08	--	4.1E-06

*Intakes adjusted based on 30% absorption of inhaled arsenic (EPA 1992b)
-- = Not Applicable

Table 3-8. Summary of Industrial Scenario Intakes Based on Maximum Contaminant Concentrations for the Horn Rapids Landfill.

Contaminant	Pathway					
	Soil Ingestion (mg/kg-d)		Fugitive Dust Inhalation (mg/kg-d)		Dermal Exposure (mg/kg-d)	
	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic
Antimony	4.5E-06	--	--	--	8.9E-08	--
Arsenic	1.9E-06	5.4E-07	--	1.1E-09*	3.7E-08	1.1E-08
Barium	3.8E-04	--	2.6E-06	NA	7.5E-06	--
Beryllium	3.7E-07	1.1E-07	--	7.2E-10	7.4E-09	2.1E-09
Chromium	3.6E-04	--	--	7.0E-07	7.1E-06	--
Copper	3.7E-04	--	--	--	7.3E-06	--
Nickel	1.6E-04	--	--	3.1E-07	3.2E-06	--
Thallium	8.9E-07	--	--	--	1.7E-08	--
Vanadium	2.9E-05	--	--	--	5.8E-07	--
Zinc	9.1E-04	--	--	--	1.8E-05	--
Beta-HCH	--	7.7E-09	--	5.2E-11	--	9.2E-09
DDT	5.7E-07	1.6E-07	--	1.1E-09	6.8E-07	1.9E-07
Heptachlor	5.8E-09	1.6E-09	--	1.1E-11	6.8E-09	1.9E-09
PCBs	--	8.4E-06	--	5.7E-08	--	1.0E-05

*Intakes adjusted based on 30% absorption of inhaled arsenic (EPA, 1992b)
-- = Not Applicable

Table 3-9. Summary of Residential Scenario Intakes Based on Maximum Contaminant Concentrations for the Soil Ingestion, Fugitive Dust Inhalation, and Dermal Exposure Pathways at Specific 1100-EM-1 Operable Subunits.

Contaminant	Pathway					
	Soil Ingestion (mg/kg-d)		Fugitive Dust Inhalation (mg/kg-d)		Dermal Exposure (mg/kg-d)	
	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic
1100-2						
Tetrachloroethene	1.3E-07	5.6E-08	--[c]	1.3E-11	1.7E-07	7.2E-08
1100-3						
Arsenic	1.3E-05	5.4E-06	--[c]	2.9E-10[a]	2.7E-07	1.1E-07
Chromium	5.2E-05	--[b]	--[c]	4.0E-09	1.1E-06	--[b]
Lead	--[c]	--[d]	--[c]	--[d]	--[c]	--[d]
UN-1100-6						
BEHP	9.3E-02	4.0E-02	--[c]	4.7E-06	1.1E-02	4.7E-03
Chlordane	6.9E-06	2.9E-06	--[c]	3.5E-10	8.8E-06	3.8E-06
Ephemeral Pool						
Chlordane	1.0E-05	4.5E-06	--[c]	1.4E-09	1.3E-05	5.7E-06
PCBs	--[c]	6.6E-05	--[c]	2.1E-08	--[c]	8.6E-05
Horn Rapids Landfill						
Arsenic	2.4E-05	1.0E-5	--[c]	2.4E-09	5.2E-07	2.2E-07
Beryllium	4.8E-06	2.1E-06	--[c]	1.5E-09	1.0E-07	4.4E-08
Chromium	4.6E-03	--[b]	--[c]	1.5E-06	9.9E-05	--[b]
Lead	--[c]	--[d]	--[c]	--[d]	--[c]	--[d]
PCBs	--[c]	1.6E-04	--[c]	1.2E-07	--[c]	2.1E-04
Tetrachloroethene	2.2E-08	9.6E-09	--[c]	7.1E-12	2.8E-08	1.2E-08

[a]Intakes adjusted for 30% absorption of inhaled arsenic (EPA, 1992b)
[b]Not considered carcinogenic by this route of exposure or pathway
[c]RfD not available to evaluate intake for this pathway.
[d]SF not available to evaluate intake for this pathway.
-- Indicates not applicable

Table 3-10. Summary of Contaminant Intakes for Homegrown Vegetables in the Garden Pathway at Specific 1100-EM-1 Operable Subunits Based on the Maximum Contaminant Concentrations in Soil.

	Leafy (lettuce)[a] (mg/kg-d)		Root (carrots)[b] (mg/kg-d)		Garden Fruits (tomatoes)[c] (mg/kg-d)		Potatoes[d] (mg/kg-d)		Total Contaminant Intake (mg/kg-d)	
	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic
1100-3										
Arsenic	2.0E-06	--[e]	8.2E-07	--[e]	2.0E-07	--[e]	2.4E-07	--[e]	3.3E-06	--[e]
Chromium	4.2E-05	--[e]	4.3E-05	--[e]	1.7E-05	--[e]	1.0E-04	--[e]	2.0E-04	--[e]
Lead	--[f]	--[g]	--[f]	--[g]	--[f]	--[g]	--[f]	--[g]	--[f]	--[g]
UN-1100-6										
BEHP	1.4E-01	6.2E-02	1.1E-01	4.7E-02	1.5E-02	6.5E-03	6.4E-02	2.6E-02	3.3E-01	1.4E-01
Chlordane	5.6E-07	2.4E-07	4.6E-05	2.0E-05	1.2E-05	5.1E-06	7.1E-05	3.0E-05	1.2E-04	5.5E-05
Ephemeral Pool										
Chlordane	8.4E-07	3.6E-07	7.0E-05	3.0E-05	1.8E-05	7.8E-06	1.0E-04	4.5E-05	1.9E-04	8.3E-05
PCBs	--[f]	1.0E-04	--[f]	7.8E-05	--[f]	1.1E-05	--[f]	4.5E-05	--[f]	2.3E-04
Horn Rapids Landfill										
Arsenic	4.0E-06	--[e]	1.6E-06	--[e]	3.9E-07	--[e]	5.1E-07	--[e]	6.4E-06	--[e]
Beryllium	8.4E-06	3.6E-06	4.1E-06	1.8E-06	1.6E-06	6.9E-07	9.9E-06	4.2E-06	2.4E-05	1.0E-05
Chromium	3.8E-03	--[e]	3.8E-03	--[e]	1.5E-03	--[e]	9.6E-03	--[e]	1.8E-02	--[e]
Lead	1.0E-04	4.4E-05	3.1E-05	1.4E-05	5.1E-05	2.2E-05	8.7E-05	3.6E-05	2.6E-04	1.2E-04
PCBs	--[f]	2.5E-04	--[f]	1.9E-04	--[f]	2.6E-05	--[f]	1.1E-04	--[f]	5.8E-04

[a]Assumes intake of 1.1 g/d dry weight (EPA, 1986a)
[b]Assumes intake of .88 g/d dry weight (EPA, 1986a)
[c]Assumes intake of 2.2 g/d dry weight (EPA, 1986a)
[d]Assumes intake of 9.1 g/d dry weight (EPA, 1986a)
[e]Not considered carcinogenic by this route of exposure or pathway
[f]RfD not available to evaluate intake for this pathway.
[g]SF not available to evaluate intake for this pathway.
-- Indicates not applicable

Table 3-11. Summary of Residential Scenario Intakes Based on the Maximum Contaminant Concentrations for the Groundwater Pathway at the Horn Rapids Landfill.

Contaminant	Pathway			
	Ingestion (mg/kg-d)		Volatile Inhalation (mg/kg-d)	
	Non-carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic
Nitrate	1.7E+00	--[a]	--[b]	--[a,b]
Trichloroethene	--[c]	1.3E-03	--[c]	4.8E-03

[a]Not considered to be a carcinogen
[b]Not a volatile contaminant
[c]RfD not available to evaluate intake for this pathway
-- Indicates not applicable

EPA is developing the UBK model to estimate blood-lead levels due to overall exposure to lead in the environment (EPA, 1991c and 1991d). The model has not officially been released, but is available from EPA for limited evaluation and use. The current model (version 0.5) estimates lead uptake and blood-lead levels in children ages 0 to 6. Children at this age are a sensitive group of individuals, are potentially more susceptible to the adverse effects associated with lead exposures.

The UBK model uses default parameters or subunit-specific information on concentrations of lead in soil, dust, air, diet, or water to predict blood-lead levels in children. The UBK model conservatively predicts absorption (uptake) of lead by various routes and applies that uptake to a compartment kinetic model. This compartment kinetic model describes lead distribution in the body and integrates the effect of The lead uptake over time. Graphical results of the UBK model are used to show the percentages of children of specific age groups that may have blood-lead levels above or below a specified concentration. For this BRSRA, a value of 10 μg/dL has been selected as the blood-lead level of interest. The Centers for Disease Control currently recommends that blood-lead levels in children should not exceed 10 μg/dL in order to prevent potential adverse intellectual development. Based on this threshold, the model's default exposure parameters results in an acceptable soil concentration of approximately 500 mg/kg.

A discussion of the application of the UBK model at the 1100-3 subunit and HRL subunit is discussed below. The results of the UBK model are discussed in chapter 5, paragraph 5.1. Computer output for the model results is provided in appendix V.

3.6.1 1100-3

The UBK model is run for two assumed residential scenarios at this subunit. First, to determine the predicted blood-lead level in children from 0 to 6 years of age, the model's default exposure parameters are used with the maximum concentration of lead detected at the 1100-3 subunit (*i.e.*, 26.4 mg/kg). This provides a subunit benchmark for each age for soil ingestion and dust inhalation based on the maximum subunit lead concentration in soil and assumes typical (default) diet, water, and air exposures.

The second scenario is to predict the blood lead level specifically in a 2-year old child (24 to 36 months) based on soil ingestion, dust inhalation, and dietary intake of lead from consumption of the four vegetable groups evaluated in the residential scenario, in conjunction with typical background. A 2-year old child has been selected because daily DW consumption for each vegetable group is readily available for this age group and young children are one of the most sensitive subpopulations for lead exposure. Additionally, this approach is consistent with the approach recommended in EPA (1986a).

To evaluate the dietary lead uptake for a 2-year old child, the daily dietary consumption of lead (as DW) via intake of subunit-grown potatoes, leafy vegetables, root vegetables, and garden fruits is determined for a 2-year old child. The concentration of lead in each of the four vegetable groups, presented in table 3-4, is multiplied by the child's DW

Table 3-12. Summary of Intakes from Residential-Related Recreational Pathways for the 1100-EM-1 Operable Unit.

Contaminant	Pathway			
	Swimming[a] (mg/kg-d)		Eating Fish (mg/kg-d)	
	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic
Trichloroethene	--[b]	1.4E-08	--[b]	3.1E-08

[a]Indicates dermally absorbed dose
[b]RfD not available to evaluate intake for this pathway

consumption of vegetables from each food group. The DW consumptions of a 2-year old child, as provided in EPA (1986a) are:

• Leafy vegetables (*e.g.*, lettuce)	0.485 g/d
• Root vegetables (*e.g.*, carrots)	0.668 g/d
• Garden fruits (*e.g.*, tomatoes)	1.669 g/d
• Potatoes	10.034 g/d

For children, potentially living at 1100-3 and consuming vegetables in these food groups that are homegrown at the subunit, the daily lead intake from these foods is estimated to be:

• Leafy vegetables (*e.g.*, lettuce)	0.1 μg/d
• Root vegetables (*e.g.*, carrots)	0.05 μg/d
• Garden fruits (*e.g.*, tomatoes)	0.009 μg/d
• Potatoes	0.21 μg/d
Total additional lead intake	0.37 μg/d

To complete this second scenario, the UBK model predicts the blood level in a 2-year old child by combining the above subunit specific total additional dietary lead intake data with the subunit specific benchmark data from the first scenario.

Model outputs for these two scenarios are discussed in chapter 5, paragraph 5.1 and provided in appendix V. The modelling results are conservative because of the assumption that all vegetables in these four food groups are homegrown and that the entire subunit is uniformly contaminated at the maximum concentration detected in the soil.

3.6.2 HRL

The UBK model was run for the same two assumed residential scenarios at HRL as were performed for 1100-3. With the exception of the concentrations in the four vegetable groups the parameters are the same as for subunit 1100-3. For children, potentially living at HRL and consuming vegetables in these food groups that are all homegrown at the subunit, the daily additional lead intake from these foods was estimated as:

• Leafy vegetables (*e.g.*, lettuce)	3.3 μg/d
• Root vegetables (*e.g.*, carrots)	1.7 μg/d
• Garden fruits (*e.g.*, tomatoes)	2.8 μg/d
• Potatoes	6.8 μg/d
Total additional lead intake	14.6 μg/d

To complete this second scenario, the UBK model predicts the blood level in a 2-year old child by combining the subunit specific total additional dietary lead intake data, with the subunit specific data from the first scenario.

Model outputs for these two scenarios are also discussed in chapter 5, paragraph 5.1 and provided in appendix V. Again, the modelling results are conservative because of the assumption that all vegetables in these four food groups are homegrown and that the entire subunit is uniformly contaminated at the maximum concentration detected in the soil.

3.7 SUMMARY OF EXPOSURE ASSESSMENT

The exposure assessment for the BISRA and BRSRA quantifies potential exposures for industrial workers who would work at a specific subunit on a regular basis and onsite residents who would live at a specific subunit, respectively. The intakes, based on both carcinogenic and noncarcinogenic effects, are estimated for exposure pathways recommended in the HSBRAM (DOE/RL-91-45, 1992) for the industrial scenario and are as requested by EPA [Einan, 1991 (see appendix I)] for the residential scenario. It has been noted that the fugitive dust intake of arsenic is reduced by 30 percent because of absorption (EPA, 1992b). Pathways determined for the BISRA are only related to soil as discussed in paragraphs 3.3 and 3.5 above. Uncertainty in the estimated exposures is discussed in chapter 5, paragraph 5.4 as part of the overall uncertainty in the characterization of risks for the 1100-EM-1 Operable Unit.

4.0 HUMAN HEALTH TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to identify the potential adverse effects associated with exposure to site-related substances and to estimate, using numerical toxicity values, the likelihood that these adverse effects may occur based on the extent of the exposure. The toxicity assessment for the BISRA was conducted in accordance with RAGS (EPA, 1989a) and is discussed in the HSBRAM (DOE/RL-91-45).

The preparation of a toxicity assessment relies primarily on existing toxicity information, and does not usually involve development of toxicity information or dose-response relationships. Current toxicological information that has already been evaluated and summarized is available in a number of documents, databases, and other sources. Toxicological profiles for the COPC are provided in appendix II.

4.1 TOXICITY INFORMATION FOR NONCARCINOGENIC EFFECTS

Systemic, toxic effects other than cancer can be associated with exposures to chemicals. The RfD is the toxicity value used to evaluate noncarcinogenic effects resulting from exposures to chemicals. The RfD has been developed based on the concept that protective mechanisms exist that must be overcome before an adverse effect is manifested (*i.e.*, there is a threshold that must be reached before adverse effects occur). The RfD is developed to reflect the duration of exposure (*e.g.*, subchronic exposures - 2 weeks to 7 years and chronic exposures - 7 years to a lifetime) and the route of exposure (*e.g.*, inhalation, oral, *etc.*). In addition, RfD's are currently being developed, as appropriate, to evaluate specific critical effects such as developmental effects that may occur because of exposure to certain chemicals.

RfD's derived from data obtained from studies in animals or humans using modification and uncertainty factors that account for uncertainty in the information used to derive the RfD. Uncertainty factors are applied for extrapolation of the no-observed-effects-level (NOEL) in a study population to the RfD used in the risk assessment. A factor of 10 is usually applied to reflect the level of each of the sources of uncertainty listed below:

- Use of lowest observed effect level (LOEL) or other parameters that are less conservative than NOEL;
- Use of data from short-term exposure studies to extrapolate to long term exposure;
- use of data from animal studies to predict human effects; and
- use of data from homogeneous animal populations or healthy human populations to predict effects in the general population.

A modifying factor may also be incorporated into the RfD to reflect qualitative professional judgements regarding scientific uncertainties not considered by the uncertainty factor, such as the completeness of the data base and the number of animals in the study. Uncertainty factors and modifying factors, as published by EPA in IRIS or HEAST, are presented in table 4-1.

For purposes of these baseline risk assessments, the chronic RfD is utilized to evaluate potential noncarcinogenic effects. The chronic RfD is a daily exposure level that is not likely to cause an appreciable lifetime risk of deleterious effects to the general population, and sensitive subpopulations.

Table 4-1 summarizes the noncarcinogenic toxicity values for the COPC at the 1100-EM-1 Operable Units evaluated. Oral RfD's have been published for all of the COPC except for PCB's and trichloroethane. Confidence in these RfD's is low or medium for all COPC except nitrate. The confidence in the RfD for nitrate is high because the values are derived from human infant studies. An inhalation RfD is published for only two of the COPC, barium and 1,1,1-trichloroethane. However, 1,1,1-trichloroethane has only been detected in soil gas (DOE/RL-90-18); and, soil gas exposures are not evaluated, as indicated in chapter 3, paragraph 3.2. . The RfD for barium is based on a 4-month inhalation study in rats that resulted in fetotoxicity. Based on this reproductive study, an interim RfD is published in HEAST, but it is under review and the RfD is subject to change.

The noncarcinogenic effects for the COPC include a variety of effects such as altered blood chemistry profiles for antimony, gastrointestinal irritation for copper, or increased blood pressure for barium. Liver effects, such as increased liver weight, lesions in the liver, or changes in liver enzymes, are associated with thallium, BEHP, chlordane, DDT, heptachlor, and tetrachloroethane. Skin effects are associated with arsenic. No critical effects are identified for beryllium or chromium by the oral route. Nitrate is associated with changes in the capacity of the blood system to transport oxygen.

Additional information on the noncarcinogenic effects for each contaminant of potential concern is provided in the toxicity profiles presented in appendix III.

4.2 TOXICITY INFORMATION FOR CARCINOGENIC EFFECTS

Toxicity values have also been developed for evaluating potential human carcinogenic effects from exposures to chemicals. Potential human carcinogenic effects are evaluated using the chemical-specific SF and accompanying EPA weight-of evidence determination. The toxicity values (*i.e.*, SF's) for carcinogens have been derived based on the premise that for any exposure to a carcinogenic chemical there is always a carcinogenic response (*i.e.*, there is no threshold). The SF is used in risk assessment to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen.

Table 4-1. Summary of Noncarcinogenic Toxicity Information for Contaminants of Potential Concern at the 1100-EM-1 Operable Unit.

Contaminant	ORAL						INHALATION					
	Oral Rfd (mg/kg-day)	Oral Rfd (basis/source)	Confidence Level	Critical Effect	Uncertainty Factors	Modifying Factors	Inhalation Rfd (mg/kg-d)	Inhalation Rfd (basis/source)	Confidence level	Critical effect	Uncertainty Factor	Modifying Factor
Antimony	4E-4	Water/IRIS	low	longevity, blood gluc.	1,000	1	--	--	--	--	--	--
Arsenic	3E-4	Food/IRIS	medium	hyperpigmentation keratosis	3	1	--	--	--	--	--	--
Barium	7E-2	Water/IRIS	medium	incr. blood press	3	1	1E-4	HEAST	--	--	1,000	--
Beryllium	5E-3	Water/IRIS	--	none observed	100	1	--	--	--	--	--	--
Chromium VI	5E-3	Water/IRIS	low	none	500	1	--	--	--	--	--	--
Copper	4E-02	EPA Region 10	--	GI irritation	--	--	--	--	--	--	--	--
Lead	ND	--	--	--	--	--	ND	--	--	--	--	--
Nickel	2E-2	Food/IRIS	medium	decrease body + organ weight	300	1	--	--	--	--	--	--
Thallium	8E-5 9E-5	-/IRIS	--	SGOT and serum LDH level	3,000	--	--	--	--	--	--	--
Vanadium	7E-3	Water/HEAST	--	none	100	--	--	--	--	--	--	--
Zinc	2E-1	HEAST	--	anemia	10	--	--	--	--	--	--	--
BEHP	2E-2	IRIS	low	liver weight	1000	1	--	--	--	--	--	--
Beta-HCH	--	--	--	--	--	--	--	--	--	--	--	--
Chlordane	6E-5	Food/IRIS	low	liver hypertrophy in mice	1,000	1	--	--	--	--	--	--
Heptachlor	5E-4	Food/IRIS	low	liver weight	300	1	--	--	--	--	--	--
PCBs	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethane	1E-2	Gavage/IRIS HEAST 1991	medium	hepatotoxic in mice, weight gain rat	1,000	1	--	--	--	--	--	--
1,1,1-Trichloroethane	9E-2	Oral/HEAST	--	--	--	--	--	--	--	--	--	--
Trichloroethane	--	--	--	--	--	--	--	--	--	--	--	--

Sources:
IRIS (Integrated Risk Information System Access): July, 1992a (EPA, 1992a)
HEAST (Health Effects Assessment Summary Tables) (EPA,1992b) unless otherwise indicated

ND = Not determined
-- Indicates not available

In addition to identifying the SF, the likelihood that a substance is a human carcinogen is also considered. A weight-of-evidence classification is assigned to each substance based on the strength of evidence of carcinogenicity. The EPA weight-of-evidence classifications are:

- Group A - Human Carcinogen
- Group B - Probable Human Carcinogen
  - B1 - Limited evidence of carcinogenicity in humans
  - B2 - Sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans
- Group C - Possible Human Carcinogen
- Group D - Not Classifiable as to Human Carcinogenicity
- Group E - Evidence of Non-Carcinogenicity in Humans

The toxicity values and supporting information for carcinogenic substances carried through the BISRA are summarized in table 4-2. EPA has not published a SF for lead in either IRIS or HEAST. Eleven of the seventeen COPC are considered carcinogens. Arsenic, chromium (VI), and nickel are known human carcinogens (EPA weight-of-evidence classification A). Arsenic is associated with lung and skin cancer by both the oral and inhalation exposure routes. Chromium (VI) and nickel are considered carcinogenic only by the inhalation route of exposure, because no evidence is currently available to indicate that they are carcinogenic by the oral route of exposure.

Beryllium, lead, BEHP, chlordane, DDT, heptachlor, and PCB's are probable human carcinogens (EPA weight-of-evidence classification B2) with insufficient human data, but sufficient data in animals to suggest that they are carcinogens. Beta-HCH is an EPA weight-of-evidence classification C, possible human carcinogen. The weight-of-evidence classification for tetrachloroethane and trichloroethane are currently under review pending resolution of the differing opinions on the classification (see appendix I).

Additional information on the toxicity associated with these COPC is provided in appendix II.

Table 4-2. Summary of Carcinogenic Toxicity Information for the Contaminants of Potential Concern at the 1100-EM-1 Operable Unit.

Contaminant	Weight of Evidence Classification	Type of Cancer	Oral SF $(mg/kg\text{-}d)^{-1}$	Oral SF (source)	Inhalation SF $(mg/kg\text{-}d)^{-1}$	Inhalation SF (source)
Arsenic	A	Skin, Lung	1.75E+00[a]	Surrogate	5.0E+01	IRIS/HEAST
Beryllium	B2	--	4.3E+00	IRIS	8.4	HEAST
Chromium VI	A	Lung	NA[b]	NA	4.1E+01	IRIS/HEAST
Lead	B2	--	ND	NA	ND	NA
Nickel	A	Lung	NA[b]	NA	8.4E-01	IRIS
BEHP	B2	--	1.4E-02	IRIS	1.4E-02[e]	Surrogate
Beta-HCH	C	--	1.8E+00	IRIS	1.8E+00	IRIS
Chlordane	B2	--	1.3E+00	IRIS	1.3E+00	IRIS
DDT	B2	--	3.4E-01	IRIS	3.4E-01	IRIS
Heptachlor	B2	--	4.5E+00	IRIS	4.5E+00	IRIS
PCBs	B2	--	7.7E+00[a]	IRIS	7.7+00[c]	Surrogate
Tetrachloroethene	B2[c,d]	--	5.2E-02	Region-10[c]	2E-03	Region-10[c]
1,1,1-Trichloroethane	NA	NA	NA	NA	NA	NA
Trichloroethane	B2[c,d]	--	1.1E-02	Region-10[c]	6.0E-03	Region-10[c]

[a]Based on proposed arsenic unit risk of 5E-05 $\mu g/L$ (EPA, 1991a)
[b]Not considered carcinogenic by oral route of exposure
[c]As recommended by Superfund Technical Support Center, April 1992 (EPA-10, see Appendix I)
[d]Weight-of-evidence classification under evaluation
[e]Surrogate; assumed same as oral SF
-- Indicates not available; presented for Class A carcinogens only
ND = Not determined
NA = Not applicable

Sources: IRIS - Integrated Risk Information Access: July, 1992 (EPA, 1992a)
HEAST - Health Effects Assessment Summary Tables (EPA, 1992b), unless otherwise indicated

This page left intentionally blank.

5.0 RISK CHARACTERIZATION

The information from the exposure assessment and the toxicity assessment is used to characterize the human health risks. The risk characterization presents quantitative and qualitative descriptions of risk. The quantification of the noncarcinogenic risk is discussed in paragraph 5.1 and quantification of carcinogenic risk is discussed in paragraph 5.2. Based on the results of the risk assessment using the maximum contaminant concentrations, contaminants that are estimated to have a risk greater than 1E-06 are considered for evaluation using the 95 percent UCL values. A discussion of the estimated risks using the 95 percent UCL is provided in paragraph 5.3. A discussion of the uncertainty in the risk characterization is provided in paragraph 5.4.

5.1 QUANTIFICATION OF NONCARCINOGENIC RISK

Potential human health hazards associated with exposure to noncarcinogenic substances, or carcinogenic substances with systemic toxicities other than cancer, are evaluated separately from carcinogenic risks. The daily intake over a specified time period (*e.g.*, lifetime or some shorter time period) is compared to an RfD for a similar time period (*e.g.*, chronic RfD or subchronic RfD) to determine a ratio called the hazard quotient (HQ). Estimates of intakes for both the BISRA and BRSRA are based on chronic exposures. The nature of the contaminant sources and the low probability for sudden releases of contaminants from the subunits preclude short-term fluctuations in contaminant concentrations that might produce acute or subchronic effects.

The formula for estimation of the HQ is:

$$HQ = \frac{\text{Daily Intake}}{\text{RfD}}$$

If the HQ exceeds unity, the possibility exists for systemic toxic effects. The HQ is not a mathematical prediction of the severity or incidence of the effects, but rather is an indication that effects may occur, especially in sensitive subpopulations. If the HQ is less than unity, then the likelihood of adverse noncarcinogenic effects is small.

RfD's are route specific. Currently, all of the RfD's in IRIS are based on ingestion and inhalation; none have been based on dermal contact. As recommended by EPA, 1992c, until more appropriate dose-response factors are available, the oral RfD's should be used to evaluate dermal exposures. EPA further recommends using the oral RfD to evaluate dermal exposures, unadjusted for absorption, unless estimates of the gastrointestinal absorption fraction are available for the compound of interest in the appropriate vehicle (EPA, 1992c). The uncertainty regarding these assumptions is discussed in paragraph 5.4.

Some contaminants do not have RfD's published in IRIS or HEAST. Therefore, the HQ cannot be calculated for these contaminants and the potential adverse effects are not evaluated. This lack of RfD's is most significant for the inhalation pathway where barium is the only contaminant of potential concern (COPC) with an inhalation RfD. It should be noted that the inhalation RfD for chromium has been withdrawn by EPA and inhalation issues are under review by the RfD/reference concentration (RfC) Work Group (EPA, 1992b). Chromium is evaluated for carcinogenicity via the inhalation pathway at subunits where it is a COPC (*e.g.*, 1100-2, 1100-3, and HRL), as discussed in paragraph 5.2.. The lack of toxicity values is discussed more fully in paragraph 5.4.

Inhalation RfD's for 1,1,1-trichloroethane have been published in IRIS or HEAST. As discussed in chapter 2 and chapter 3, paragraph 3.2; 1,1,1-trichloroethane has only been detected in soil gas and groundwater at low concentrations and has not been quantitatively evaluated. Consequently, the HQ's are not determined for any of the COPC in the inhalation pathway. All COPC have published SF's, used to estimate carcinogenic risk. Carcinogenic effects usually occur at levels significantly lower than those associated with systemic toxic effects; therefore, cancer is usually the predominant adverse effect for contaminants that produce carcinogenic as well as systemic toxic effects.

The HQ for all contaminants for a specific pathway or a scenario can be summed to provide a hazard index (HI) for that pathway or scenario.

Lead exposures have been evaluated for potential adverse impacts using the UBK Model. The results are presented in appendix V and summarized below for 1100-3 BRSRA and the HRL BRSRA.

5.1.1 SUMMARY OF SYSTEMIC TOXIC EFFECTS FOR BISRA

5.1.1.1 1100-1 BISRA. The noncarcinogenic HQ's for the COPC are presented in table 5-1. The HI for the soil ingestion pathway and dermal exposure pathways is at least 2 orders of magnitude less than unity. Therefore, adverse systemic toxic health effects in industrial workers are not likely from ingestion or dermal exposure to the maximum concentrations of arsenic or vanadium detected at this subunit. Neither arsenic nor vanadium have inhalation RfD's; therefore, an HI is not calculated for this exposure pathway. Arsenic is evaluated for carcinogenicity via inhalation in paragraph 5.2.

5.1.1.2 1100-2 BISRA. Chromium is the only COPC at this subunit. The overall hazard index for exposure to chromium through the ingestion or dermal route of exposure is 0.001 as presented in table 5-1. Therefore, adverse systemic toxic health effects are not likely for industrial workers exposed to the COPC at 1100-2 via the ingestion or dermal pathways.

5.1.1.3 1100-3 BISRA. Chromium is the only COPC at this subunit. The noncarcinogenic HQ's for chromium are presented in table 5-1. The HI for the soil ingestion and dermal pathway is 0.0008. Adverse systemic toxic effects in industrial workers are not likely from ingestion or dermal exposure to chromium detected in soils at this subunit.

5.1.1.4 1100-4 BISRA. The noncarcinogenic HQ's for arsenic and beryllium, the two COPC at this subunit, are presented in table 5-1. As indicated in chapter 3, paragraph 3.3.1, the contaminated soils are located beneath a cement floor in a building, and hence the fugitive dust pathway is not evaluated. The quantitative evaluation of the soil ingestion and dermal pathways is provided for information purposes. Both the soil ingestion HI and the dermal exposure pathway HI are at least 2 orders of magnitude less than unity. If contact with the COPC were to occur by soil ingestion and dermal exposure, adverse systemic toxic effects in industrial workers are not likely.

5.1.1.5 UN-1100-6 Subunit BISRA. The HQ's for the COPC at this subunit are also presented in table 5-1. The HI for the soil ingestion pathway is 0.4 and is due primarily to the BEHP present in the soil at this site. The HI for the dermal exposure pathway is 0.05. Adverse systemic toxic effects in industrial workers are not likely from ingestion or dermal exposure to COPC detected at this subunit. None of the COPC have published inhalation RfD's, so no inhalation HQ's or HI's are presented, although they have all been evaluated for carcinogenity in paragraph 5.2.

5.1.1.6 Ephemeral Pool BISRA. The HQ's for the COPC at the Ephemeral Pool are presented in table 5-1. The soil ingestion HI is 0.01 and the dermal exposure pathway HI is 0.02. Adverse systemic toxic effects in industrial workers are not likely for soil ingestion or dermal exposure to COPC detected at this subunit. None of the COPC have published inhalation RfD's, so no inhalation HQ's or HI's are presented, although they have all been evaluated for carcinogenity in paragraph 5.2.

5.1.1.7 HRL BISRA. The HQ's for the COPC at HRL are presented in table 5-2. The soil ingestion pathway HI is 0.2, the fugitive dust inhalation pathway HI is 0.03, and the dermal exposure pathway HI is 0.003. The total subunit HI is 0.2. Consequently, adverse systemic toxic effects are not likely from potential exposures to the COPC for industrial workers at this subunit.

5.1.2 SUMMARY OF SYSTEMIC TOXIC EFFECTS FOR BRSRA

5.1.2.1 1100-2 BRSRA. Tetrachloroethene is the only COPC at 1100-2. The individual HQ and overall HI for exposure to tetrachloroethene through the ingestion or dermal route of exposure is 5 orders of magnitude less than unity, as presented in table 5-3. The garden produce exposure pathway assessment for each subunit is presented in table 5-4, with the exception of 1100-2, because tetrachloroethene is not evaluated for the garden pathway. Adverse systemic toxic effects are unlikely for residents who may be exposed to tetrachloroethene in the soil at this subunit. As presented in table 5-7, HI estimated for this subunit is 0.00003.

5.1.2.2 1100-3 BRSRA. The HQ's via the ingestion and dermal pathways for the COPC at this subunit are presented in table 5-3. For soil ingestion and dermal exposure, the HQ's are all less than unity. The HI for the soil ingestion pathway is 0.05 and for the dermal exposure pathway is 0.001. Adverse systemic toxic effects in residents are not likely from exposure to the arsenic and chromium detected in soils at this subunit.

Table 5-1. Summary of the Baseline Industrial Scenario Risk Assessment for 1100-1, 1100-2, 1100-3, 1100-4, UN-1100-6, and the Ephemeral Pool. (Sheet 1 of 2)

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HI[c]	ICR[b]
1100-1										
Arsenic	0.003	4E-07	--	9E-12[d]	0.00006	9E-09	0.003	4E-07		
Vanadium	0.005	--	--	--	0.0001	--	0.005	--		
Pathway Totals	0.008	4E-07	--	9E-12	0.0002	9E-09			0.008	4E-07
1100-2										
Chromium	0.001	--	--	1E-07	0.00002	--	0.001	1E-07	0.001	1E-07
1100-3										
Chromium	0.0008	--	--	8E-08	0.00002	--	0.0008	8E-08	0.0008	8E-08
1100-4										
Arsenic	0.006	8E-07	--	--	0.0001	2E-08	0.006	8E-07		
Beryllium	0.0005	3E-07	--	--	0.000001	6E-09	0.0005	3E-07		
Pathway Totals	0.006	1E-06	--	--	0.0001	3E-08			0.006	1E-06
UN-1100-6										
BEHP	0.4	3E-05	--	3E-08	0.04	3E-06	0.4	3E-05		
Chlordane	0.009	2E-07	--	2E-10	0.01	2E-07	0.02	4E-07		
Heptachlor	0.0004	2E-08	--	3E-10	0.00004	3E-08	0.0004	5E-08		
Pathway Totals	0.4	3E-05	--	3E-08	0.05	3E-06			0.4	3E-05

Table 5-1. Summary of the Baseline Industrial Scenario Risk Assessment for 1100-1, 1100-2, 1100-3, 1100-4, UN-1100-6, and the Ephemeral Pool. (Sheet 2 of 2)

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HI[c]	ICR[b]
Ephemeral Pool										
Chlordane	0.01	3E-07	--	9E-10	0.02	3E-07	0.03	6E-07		
Heptachlor	0.00002	1E-08	--	3E-11	0.00002	1E-08	0.00004	2E-08		
PCBs	--	3E-05	--	8E-08	--	3E-05	--	6E-05		
Pathway Totals	0.01	3E-05	--	8E-08	0.02	3E-05			0.03	6E-05

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Hazard Index
[d]Based on 30% absorption of inhaled arsenic (EPA 1992b)
-- = Not Applicable

Table 5-2. Summary of the Baseline Industrial Scenario Risk Assessment Based on Maximum Contaminant Concentrations for the Horn Rapids Landfill.

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HI[c]	ICR[b]
Antimony	0.01	--	--	--	0.0004	--	0.01	--		
Arsenic	0.006	9E-07	--	6E-08[d]	0.0001	2E-08	0.006	1E-06		
Barium	0.005	--	0.03	--	0.00001	--	0.04	--		
Beryllium	0.00007	5E-07	--	6E-09	0.000002	9E-09	0.00007	5E-07		
Chromium	0.07	--	--	3E-05	0.001	--	0.07	3E-05		
Copper	0.009	--	--	--	0.0002	--	0.009	--		
Nickel	0.008	--	--	3E-07	0.0002	--	0.008	3E-07		
Thallium	0.01	--	--	--	0.0002	--	0.01	--		
Vanadium	0.004	--	--	--	0.00008	--	0.004	--		
Zinc	0.05	--	--	--	0.0009	--	0.05	--		
Beta-HCH	--	1E-08	--	9E-11	--	2E-08	--	3E-08		
DDT	0.001	5E-08	--	4E-10	0.001	6E-08	0.002	1E-07		
Heptachlor	0.00001	7E-09	--	5E-11	0.00001	9E-09	0.00002	2E-08		
PCBs	--	6E-05	--	4E-07	--	8E-05	--	1E-04		
Pathway Totals	0.2	6E-05	0.03	3E-05	0.003	8E-05			0.2	2E-04

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Hazard Index
[d]Based on 30% absorption of inhaled arsenic (EPA 1992b)
-- = Not Applicable

Table 5-1. Summary of Baseline Residental Scenario Risk Assessment Based on Maximum Contaminant Concentrations for the Soil Ingestion, Fugitive Dust Inhalation, and Dermal Exposure Pathways, for Specific 1100-EM-1 Operable Subunits. (Sheet 1 of 2)

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[b]	ICR[b]	HI[c]	ICR[b]
1100-2										
Tetrachloroethene	0.00001	3E-09	--[e]	3E-14	0.00002	4E-09	0.00003	7E-09	0.00003	7E-09
1100-3										
Arsenic	0.04	9E-06	--[e]	1E-08[d]	0.0009	2E-07	0.04	9E-06		
Chromium	0.01	--[f]	--[e]	2E-07	0.0002	--[f]	0.01	2E-07		
Lead	--[e]	--[g]	--[e]	--[g]	--[e]	--[g]	ND	ND		
Pathway Totals	0.05	9E-06	--[e]	2E-07	0.001	2E-07			0.05	9E-06
UN-1100-6										
BEHP	4.6	6E-04	--[e]	7E-08	0.5	7E-05	5.1	7E-04		
Chlordane	0.1	4E-06	--[e]	5E-10	0.2	5E-06	0.3	9E-06		
Pathway Totals	4.7	6E-04	--[e]	7E-08	0.7	8E-05			5.4	7E-04
Ephemeral Pool										
Chlordane	0.2	4E-06	--[e]	2E-09	0.2	7E-06	0.4	1E-05		
PCBs	--[e]	5E-04	--[e]	2E-07	--[e]	7E-04	--[e]	1E-03		
Pathway Totals	0.2	5E-04	--[e]	2E-07	0.2	7E-04			0.4	5E-04

Table 5-1. Summary of Baseline Residental Scenario Risk Assessment Based on Maximum Contaminant Concentrations for the Soil Ingestion, Fugitive Dust Inhalation, and Dermal Exposure Pathways, for Specific 1100-EM-1 Operable Subunits. (Sheet 2 of 2)

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[b]	ICR[b]	HI[c]	ICR[b]
Horn Rapids Landfill										
Arsenic	0.08	2E-05	--[e]	1E-07[d]	0.002	4E-07	0.08	2E-05		
Beryllium	0.001	9E-06	--[e]	1E-08	0.00002	2E-07	0.001	9E-06		
Chromium	0.9	--[f]	--[e]	6E-05	0.02	--[f]	0.9	6E-05		
Lead	--[e]	--[g]	--[e]	--[g]	--[e]	--[g]	ND	ND		
PCBs	--[e]	1E-03	--[e]	9E-07	--[e]	2E-03	--[e]	3E-03		
Tetrachloroethene	0.000002	5E-10	--[e]	1E-14	0.000003	6E-10	0.000005	1E-09		
Pathway Totals	1	1E-03	--[e]	6E-05	0.02	2E-03			1	3E-03

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Hazard Index
[d]Based on 30% absorption of inhaled arsenic (EPA, 1992b)
[e]RfD not available to evaluate this pathway
[f]Not considered carcinogenic by this route of exposure
[g]SF not available to evaluate this pathway
ND Not determined
-- Indicates not applicable

Table 5-4. Summary of Baseline Residential Scenario Risk Assessment for the Garden Pathway at Specific 1100-EM-1 Operable Subunits Based on Maximum Contaminant Concentration.

Contaminant	Pathway	
	Garden	
	HQ[b]	ICR[a]
1100-3		
Arsenic	0.01	--[d]
Chromium	0.04	--[d]
Lead	--[e]	--[f]
Total Pathway ICR	--	--
Total Pathway HI[c]	0.05	--
UN-1100-6		
BEHP	16.0	2E-03
Chlordane	2.0	7E-05
Total Pathway ICR	--	2E-03
Total Pathway HI[c]	18	--
Ephemeral Pool		
Chlordane	3.2	1E-04
PCBs	--[e]	2E-03
Total Pathway ICR	--	2E-03
Total Pathway HI[c]	3.2	--
Horn Rapids Landfill		
Arsenic	0.02	--[d]
Beryllium	0.005	4E-05
Chromium	3.6	--[d]
PCBs	--[e]	4E-03
Lead	--[e]	--[f]
Total Pathway ICR	--	4E-03
Total Pathway HI[c]	3.6	--

[a]Lifetime Incremental Cancer Risk
[b]Hazard Quotient
[c]Hazard Index
[d]Not considered carcinogenic by this route of exposure
[e]RfD not available to evaluate this pathway
[f]SF not available to evaluate this pathway
-- Indicates not applicable

For the garden produce exposure pathway, the overall HI is 0.05, as presented in table 5-4. Adverse health effects from exposure to residents though this pathway are not likely.

The results of the UBK model for evaluating residential exposures for children indicate that adverse health effects from the exposure to lead, which was detected at the relatively low concentration of 26.4 mg/kg, would not occur. Based on soil ingestion, dust inhalation, and the ingestion of garden produce grown at the subunit, blood-lead levels are not likely to exceed 10 μg/dl for a 2-year old child (see appendix V).

5.1.2.3 UN-1100-6 Subunit BRSRA. The HQ's for the COPC at this subunit are presented in tables 5-3 and 5-4. The HI for the soil ingestion pathway is 4.7 and is almost entirely due to the BEHP present in the soil at this site. The HI for the dermal exposure pathway is 0.7. Adverse systemic toxic effects in residents may occur if there was exposure to the COPC detected at this subunit. None of the COPC have published inhalation RfD's, so HQ's and HI's are not presented for the inhalation pathway.

For the garden produce exposure pathway, the overall HI is 18. As presented in table 5-4, the HI is based on a HQ of 16 for BEHP and a HQ of 2 for chlordane. The garden pathway risk is calculated by using a surrogate uptake factor for BEHP based on PCB's. As summarized in table 5-7, the subunit HI for all pathways evaluated is 23.

5.1.2.4 Ephemeral Pool BRSRA. The HQ's for the COPC at the Ephemeral Pool are presented in tables 5-3 and 5-4. The soil ingestion HI is 0.2 and the dermal exposure pathway HI is 0.2. Adverse systemic toxic effects in residents for these two exposure routes are not likely for the COPC detected at this subunit.

RfD's are not published for PCB's for the oral, inhalation, or dermal routes of exposure. Thus, the pathway and subunit HI's may be an underestimation of the likelihood of adverse systemic toxic effects because PCB's are not included in the overall evaluation.

The HQ's and HI for the garden produce exposure pathway are presented in table 5-4. The HI for the garden pathway is 3.2 and is due entirely to the chlordane detected on the site. PCB's are not quantitatively evaluated because there is no published oral RfD for PCB's. As summarized in table 5-7, the subunit HI is 3.6 indicating a potential for adverse effects based on the assumptions used for the residential scenario.

5.1.2.5 HRL BRSRA. The HQ's for the COPC at HRL are presented in tables 5-3 through 5-6. The soil ingestion HI is 1 and the dermal exposure pathway HI is 0.02. The HI of 1 for the soil ingestion pathway is due primarily to the potential exposure to chromium in the soil. Although the HI for the soil ingestion pathway is 1, adverse systemic toxic effects in residents are not likely because of the conservative assumptions used to assess potential exposures. For example, it is assumed that exposure to the maximum concentration detected in the soil occurs. Based on the spatial distribution for chromium presented in DOE/RL-90-18, only a very small area of the soil may be contaminated with the maximum concentration.

For the garden produce pathway for residents at HRL, the estimated HI is 3.6. Chromium, with an HQ of 3.6, contributes the most to this value. Arsenic and beryllium all have HQ's much less than unity. PCB's and lead do not have published RfD's for the evaluation of oral ingestion. Thus, the pathway and subunit HI's may be an underestimation of the likelihood of adverse effects. However, it should be noted that PCB's are evaluated for carcinogenic effects (see paragraph 5.2). Lead is evaluated for adverse effects using the UBK model as discussed below. As discussed in chapter 3, paragraph 3.3.1, tetrachloroethene, although detected in soil, has a limited potential for uptake or accumulation in plants. Similarly, trichloroethene and 1,1,1-trichloroethane, detected in soil gas only, are not quantitatively evaluated for this pathway, and are not presented in table
5-4.

None of the COPC except 1,1,1-trichloroethane have published RfD's for the inhalation route of exposure. Therefore, neither HQ's nor HI's are provided for this potential exposure.

The results of the UBK model for evaluating residential exposures to lead for children are presented in appendix V. For the first scenario, based on the default model parameters and using the maximum concentration of lead detected in the soil at HRL (854 mg/kg), the geometric mean for a blood-lead level in 2-year old children (24 to 36 months) is predicted to be 5.8 μg/dl. As indicated in the graph presented in appendix V for the default parameters, based on the maximum concentration of lead detected at HRL and conservative UBK model parameters, the geometric mean and geometric standard deviation of the predicted blood-lead levels indicates that approximately 5 percent of the exposed children would be expected to have a blood-lead level greater than 10 μg/dl.

When the ingestion of lead, through the consumption of homegrown vegetables, is added to other intakes evaluated in this exposure pathway, the geometric mean for the blood-lead level in a 2-year old child is predicted to be 7.01 μg/dl. As indicated in the graph presented in appendix V for the default parameters, based on the maximum concentration of lead detected at HRL and conservative UBK model parameters, the geometric mean and geometric standard deviation indicates that approximately 14 percent of the 2-year old children would be expected to have a blood-lead level greater than 10 μg/dl. However, because of the conservatism in the analysis using the maximum concentration, the actual exposure is unlikely to produce toxic effects.

The results of the risk assessment for two contaminants detected in groundwater in the vicinity of HRL are presented in table 5-5. The HQ for nitrate, based on groundwater ingestion, is 1. No oral RfD is available for evaluating trichloroethene; therefore, a HQ is not presented for this compound. This may result in an underestimation of the HI for the groundwater pathway. As shown in table 5-7, the HI for HRL is 5.6.

Table 5-5. Summary of Baseline Residential Scenario Risk Assessment Based on the Maximum Contaminant Concentrations for the Groundwater Pathway

Contaminant	Pathway			
	Groundwater Ingestion		Groundwater Inhalation	
	HQ[a]	ICR[b]	HQ[a]	ICR[b]
Nitrate	1	--[c]	--[d]	--[c,d]
Trichloroethene	--[e]	1E-05	--[e]	3E-05

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Not considered to be a carcinogen
[d]Not a volatile contaminant
[e]RfD not available to evaluate this pathway
-- Indicates not applicable

Table 5-6. Summary of Baseline Risk Assessment for the Residential-Related Recreational Pathways for the 1100-EM-1 Operable Unit.

Contaminant	Pathway			
	Swimming		Eating Fish	
	HQ[a]	ICR[b]	HQ[a]	ICR[b]
Trichloroethene	--[c]	1E-10	--[c]	3E-10

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]RfD not available to evaluate this pathway
-- Indicates not applicable

Table 5-7. Summary of the Baseline Residential Scenario Risk Assessment for Specific 1100-EM-1 Operable Subunits Based on Maximum Contaminant Concentrations.

Subunit	Pathway	Pathway Totals		Subunit Totals	
		HI[a]	ICR[b]	HI[a]	ICR[b]
1100-2	Soil Ingestion	0.00001	3E-09		
	Fugitive Dust Inhalation	--	3E-14		
	Dermal Exposure	0.00002	4E-09		
	Garden Produce	--	--		
				0.00003	7E-09
1100-3	Soil Ingestion	0.05	9E-06		
	Fugitive Dust Inhalation	--	2E-07		
	Dermal Exposure	0.001	2E-07		
	Garden Produce	0.05	--		
				0.1	9E-06
UN-1100-6	Soil Ingestion	4.7	6E-04		
	Fugitive Dust Inhalation	--	7E-08		
	Dermal Exposure	0.7	8E-05		
	Garden Produce	18	2E-03		
				23	3E-03
Ephemeral Pool	Soil Ingestion	0.2	5E-04		
	Fugitive Dust Inhalation	--	2E-07		
	Dermal Exposure	0.2	7E-04		
	Garden Produce	3.2	2E-03		
				3.6	3E-03
Horn Rapids Landfill	Soil Ingestion	1	1E-03		
	Fugitive Dust Inhalation	--	6E-05		
	Dermal Exposure	0.02	2E-03		
	Garden Produce	3.6	4E-03		
	Groundwater Ingestion	1	1E-05		
	Inhalation of Volatiles from Groundwater	--	3E-05		
				5.6	7E-03
Recreational	Dermal Exposure while swimming	--	1E-10		
	Ingestion of Fish	--	3E-10		
				--	4E-10

[a]Hazard Index
[b]Lifetime Incremental Cancer Risk
-- Indicates not applicable

5.1.2.6 Residential-Related Recreational Activities. As discussed in paragraph 3.2, modeling presented in DOE/RL 90-18 indicates that nitrate and trichloroethene, currently found in the groundwater in the vicinity of the 1100-EM-1 Operable Unit, would enter the Columbia River at concentrations less than their respective MCL's. Neither an oral nor a dermal RfD is published for trichloroethene. Trichloroethene, however, is evaluated quantitatively for potential carcinogenic effects, as discussed in paragraph 5.2. Nitrogen in the form of dissolved nitrate is an essential nutrient and does not bioaccumulate. Therefore, as summarized in tables 5-6 and 5-7, HQ's to evaluate noncarcinogenic adverse effects from potential exposures to nitrate or trichloroethene by subunit residents, who may swim in the Columbia River, or eat fish from the Columbia River are not calculated.

5.2 QUANTIFICATION OF CARCINOGENIC RISK

For carcinogens, risks are estimated as the likelihood of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen (*i.e.*, incremental or excess ICR). The equation for risk estimation is:

$$\text{ICR} = (\text{Chronic Daily Intake}) (\text{Slope Factor})$$

This linear equation is only valid at low-risk levels (*i.e.*, below estimated risks of 1E-02), and is an upperbound estimate of the upper 95th percent confidence limit of the slope of the dose-response curve. Thus, one can be reasonably confident that the actual risk is likely to be less than that predicted. Cancer risk estimates are expressed using one significant figure only.

Contaminant-specific ICR's are assumed to be additive so that ICR's can be summed for pathways and contaminants to provide pathway, contaminant, or subunit ICR's.

ICR's are presented for those contaminants known to be carcinogenic by a specific route of exposure. For example, chromium is only carcinogenic by the inhalation route of table 5-2 exposure. Consequently, an ICR is presented only for the exposure to chromium through the inhalation of fugitive dust. All COPC that are classified as human carcinogens, or probable human carcinogens, have published inhalation and oral SF's with two exceptions:

- PCB's and BEHP do not have a published inhalation SF. For purposes of this BISRA, the inhalation SF is assumed to be the same as the oral SF.

- No SF's are published for lead. Therefore, this contaminant of interest is not evaluated for its potential contribution to the subunit total ICR. This may result in an underestimation of the ICR for a subunit. The potential exposures to lead are discussed in paragraph 5.4.

As discussed above in paragraph 5.1, all of the toxicity factors in IRIS are based on ingestion and inhalation. None of the toxicity factors have been based on dermal contact. As recommended by EPA (1992c), until more appropriate dose-response factors are

available, the oral SF's should be used to evaluate dermal exposures. The EPA further recommends using the oral SF unadjusted for absorption, unless estimates of the gastrointestinal absorption fraction are available for the compound of interest in the appropriate vehicle (EPA 1992c). For the BISRA and BRSRA, the oral SF's have not been modified for absorption efficiencies. The uncertainty regarding this assumption is discussed in paragraph 5.4.

Arsenic is approximately 30 percent absorbed when inhaled (EPA, 1991). The SF for arsenic is based on an absorbed intake, therefore, all intakes are adjusted by 30 percent to calculate the risks for arsenic inhalation exposures at 1100-1, 1100-4, and HRL.

The results of the risk characterization for carcinogenic effects are presented below by subunit and summarized in tables 5-1 and 5-2. These risk estimates are based on the maximum detected contaminant concentrations. EPA considers a 1E-06 risk level to be the point of departure for determining remediation goals for alternatives when applicable or relevant and appropriate requirements (ARAR's) are not available or not sufficiently protective [40 CFR §300.430(e)(2)(i)(A)(2)].

5.2.1 SUMMARY OF CARCINOGENIC RISK FOR BISRA

5.2.1.1 1100-1 BISRA. The carcinogenic risk estimates for this subunit are presented in table 5-1. The estimated ICR for the soil ingestion pathway is 4E-07, for the fugitive dust inhalation pathway it is 9E-12, and for the dermal exposure pathway it is 9E-09. The total subunit ICR is estimated at 4E-07. Potential ingestion of arsenic contaminated soil contributes solely to this estimated risk.

5.2.1.2 1100--2 BISRA. Chromium is the only COPC at the 1100-2 subunit. Chromium, as chromium(VI), is carcinogenic only by the inhalation route of exposure. The inhalation pathway ICR for chromium, and then the total ICR, at this subunit is 1E-07 (see table 5-1). This ICR may be an overestimate of the risk because it is conservatively assumed that all chromium present in the soil is chromium(VI) and that the entire subunit is uniformly contaminated at the maximum concentration detected.

5.2.1.3 1100-3 BISRA. Again, chromium is the only COPC at this subunit. The estimated ICR's associated with chromium exposure at this subunit is presented in table 5-1. The estimated ICR for the inhalation pathway and the total subunit ICR is 8E-08.

5.2.1.4 1100-4 BISRA. The estimated ICR's associated with exposures at 1100-4 are presented in table 5-1. Arsenic and beryllium are the only two COPC at this subunit. The ICR associated with the ingestion pathway is 8E-07 for arsenic and 3E-07 for beryllium. The dermal exposure pathway also results in negligible ICR's of 2E-08 for arsenic and 6E-09 for beryllium. The inhalation of fugitive dust is not evaluated, as discussed previously, because of the location of the contamination. The ICR for the scenario (*i.e.*, sum of all individual contaminant and pathway ICR's) is negligible (*i.e.*, 1E-06). Actual risk would be much less than the estimated risks because of the limited area of this subunit, the use of the

maximum concentration of the COPC to calculate the ICR's, and the location of the contamination beneath a cement floor in an existing building.

5.2.1.5 UN-1100-6 Subunit BISRA. The estimated ICR's associated with the three exposure pathways at UN-1100-6 subunit are presented in table 5-1. The soil ingestion pathway ICR is 3E-05, the inhalation pathway ICR is 3E-08, and the dermal exposure pathway is 3E-06. All of the ICR's are associated primarily with potential exposures to BEHP at the high concentrations detected in the soil at this subunit. All other carcinogenic COPC are associated with negligible risks. The estimated subunit ICR for UN-1100-6 subunit is 3E-05, is due primarily to BEHP.

5.2.1.6 Ephemeral Pool BISRA. The estimated ICR's associated with the COPC at the Ephemeral Pool are presented in table 5-1. The soil ingestion pathway ICR is 3E-05; the fugitive dust inhalation pathway is 8E-08; and the dermal exposure pathway is 3E-05. The ICR's are due primarily to potential exposures to PCB's. ICR's for chlordane and heptachlor are less than 1E-06. The subunit ICR for all pathways is 6E-05.

5.2.1.7 HRL BISRA. The summary of ICR's for HRL, based on the maximum detected contaminant concentrations, is presented in table 5-2. The pathway-specific ICR for soil ingestion is 6E-05. PCB's detected in the soil have an ICR of 6E-05 for this pathway and are the primary COPC for this pathway. All other COPC are estimated to have negligible cancer risks by the soil ingestion pathway.

The fugitive dust pathway at HRL is estimated to have an ICR of 3E-05 due primarily to the potential exposure to chromium. This ICR may be an overestimate of the actual risk because it is conservatively assumed that all chromium present in the soil is chromium(VI) and that the entire subunit is contaminated at the maximum concentration detected. Under natural soil conditions, organic matter in the soil stimulates the reduction of chromium(VI) to the more stable, less mobile trivalent chromium (Kabata-Pendias and Pendias, 1984). As indicated in the Phase I RI report (DOE/RL-90-18), this maximum chromium concentration has been detected in only a single sample of HRL soil at a depth of 14.6 to 16.9 ft with the maximum concentration elsewhere less than 300 mg/kg and most detections less than 50 mg/kg.

For the other COPC, the ICR's for the inhalation pathway are all less than 1E-06. As with chromium, these risks are also likely to be overestimates because it is assumed that the entire landfill area is contaminated at the maximum concentration detected for each COPC.

The pathway-specific ICR for the dermal exposure pathway is 8E-05 and is associated primarily with PCB's. All other carcinogenic COPC are associated with low cancer risks by the dermal exposure pathway.

Contaminant-specific ICR's that are equal to or exceed 1E-06 at HRL are 1E-06 for arsenic, 3E-05 for chromium, and 1E-04 for PCB's.

5.2.2 SUMMARY OF CARCINOGENIC RISK FOR BRSRA

5.2.2.1 1100-2 BRSRA. Tetrachloroethene is the only COPC at the 1100-2 subunit. The ICR for the subunit, based on soil ingestion, inhalation of fugitive dust, and dermal exposures, is 7E-09 (table 5-3). As discussed in chapter 3, paragraph 3.2 and summarized in table
5-4, tetrachloroethene is not a COPC through the garden produce pathway and an ICR has not been calculated for this exposure pathway. As summarized in table 5-7, the subunit ICR is also 7E-09.

5.2.2.2 1100-3 BRSRA. The estimated ICR's associated with soil ingestion, fugitive dust inhalation, and dermal exposures at this subunit are presented in table 5-3. Chromium is only evaluated for the inhalation pathway because it is not known to be carcinogenic by other routes of exposure as discussed in chapter 4, paragraph 4.2. The estimated ICR for the soil ingestion pathway is 9E-06 and is primarily associated with arsenic detected in the soil. The ICR for arsenic is 1E-08 and for chromium is 2E-07. The estimated ICR for the inhalation pathway is 2E-07. The estimated ICR for the dermal exposure pathway is 2E-07. For these three exposure pathways, the subunit ICR is 9E-06.

For the garden produce pathway (see table 5-4), an ICR is not presented. As discussed in chapter 4, paragraph 4.2, arsenic in plants is usually the less toxic organic form that is noncarcinogenic and there is no evidence that chromium is carcinogenic by the oral route of exposure. SF's are not published for evaluating the potential carcinogenicity of lead. This could result in an underestimation of the overall subunit ICR. However, the very low concentrations of lead detected in the soil suggest lead exposures would be very low.

5.2.2.3 UN-1100-6 Subunit BRSRA. The estimated ICR's associated with the exposure pathways at UN-1100-6 subunit are presented in tables 5-3 and 5-4. The soil ingestion pathway ICR is 6E-04, the inhalation pathway ICR is 7E-08, and the dermal exposure pathway is 8E-05. Potential exposures to BEHP at the concentrations detected in the soil at this subunit yield the greatest estimated ICR. Chlordane ICR's for the soil ingestion and dermal exposure pathways are also both greater than 1E-06.

For the garden produce pathway at this subunit, the total ICR is 2E-03. BEHP is estimated to have an ICR of 2E-03 and chlordane is estimated to have an ICR of 7E-05, as presented in table 5-4.

Further discussion of the risks estimated for BEHP and chlordane is provided in paragraph 5.3. As summarized in table 5-7, the subunit ICR is 3E-03.

5.2.2.4 Ephemeral Pool BRSRA. The estimated ICR's associated with the COPC at the Ephemeral Pool are presented in tables 5-3 and 5-4. The soil ingestion pathway ICR is 5E-04, the fugitive dust inhalation pathway is 2E-07, and the dermal exposure pathway is 7E-04. The ICR's are primarily the result of potential exposures to PCB's. However, chlordane is also estimated to have an ICR of 4E-06 via soil ingestion and 7E-06 via dermal exposure to soil.

For the garden produce pathway, the total ICR for the Ephemeral Pool is 2E-03. PCB's are associated with an estimated ICR of 2E-03 and chlordane is estimated to have an ICR of 1E-04.

Further discussion of the risks estimated for PCB's and chlordane detected at the Ephemeral Pool is provided in paragraph 5.3. As summarized in table 5-7, the subunit ICR, based on the maximum contaminant concentrations, is 3E-03.

5.2.2.5 HRL BRSRA. The summary of ICR's for HRL is presented in tables 5-3 through 5-6. The pathway-specific ICR for soil ingestion is 1E-03. PCB's detected in the soil contribute most of this risk. However, arsenic and beryllium are also associated with individual ICR's that exceed 1E-06.

The fugitive dust pathway at HRL is estimated to have an ICR of 6E-05, based on the potential exposure to chromium. This ICR may be an overestimate of the risk, as discussed above for BISRA.

The pathway-specific ICR for the dermal exposure pathway is 2E-03 and is associated primarily with PCB's. All other carcinogenic COPC are associated with low cancer risks for the dermal exposure pathway.

The estimated ICR's for the garden produce pathway at this subunit is presented in table 5-4. The total pathway ICR is 4E-03. Individual COPC ICR's are beryllium (4E-05) and PCB's (4E-03).

As summarized in table 5-5, trichloroethene detected in groundwater are evaluated for exposure through ingestion of groundwater and the inhalation of volatiles from groundwater use in a residence. Using the maximum concentration of trichloroethene detected, the ICR for groundwater ingestion is 1E-05. The risk due to inhalation of volatile trichloroethene from groundwater use is 3E-05. As summarized in table 5-7, the subunit total ICR is 7E-03 with the garden produce pathway contributing an ICR of 4E-03.

5.2.2.6 Residential-Related Recreational Pathways. Trichloroethene in groundwater may be transported to the Columbia River based on modeling presented in the Phase II RI (DOE/RL-90-18). The estimated ICR's for residents who may be exposed to trichloroethene through swimming in the Columbia River or ingesting fish from the Columbia River are 1E-10 and 3E-10, respectively, as presented in tables 5-6 and 5-7.

5.3 RISK CHARACTERIZATION BASED ON THE 95 PERCENT UCL

The HQ's and ICR's presented in paragraphs 5.2 and 5.3 are based on the maximum concentration of the contaminant detected in the soil or groundwater. Several COPC at 1100-3, UN-1100-6 subunit, the Ephemeral Pool, and HRL are all associated with ICR's estimated to be greater than 1E-06. Additional evaluation of these COPC detected at these sites was conducted to provide additional characterization of the risk. None of the contaminants evaluated at any of the 1100-EM-1 operable subunits are estimated to have

exposures resulting in HQ's that exceed 1 for the industrial or residential scenario based on the maximum detected contaminant concentration. Therefore, discussion of HQ's is not provided below, although the HQ's based on the 95 percent UCL of the mean contaminant concentration are presented. The results of the evaluation of potential risks related to cancer (*i.e.*, ICR's) are discussed in this paragraph for each subunit or both the industrial and residential scenarios.

The sampling data from both the Phase I and Phase II site investigations were used to calculate the 95 percent UCL of the mean contaminant concentration at these subunits. The procedure and data used to calculate the 95 percent UCL are presented in appendix IV. Data was used to calculate the 95 percent UCL that best represented the spatial distribution of contaminants. This provides a conservative estimate of the mean concentrations since low values and nondefects are not used. The 95 percent UCL is used to estimate contaminant intakes, HQ's, and ICR's.

5.3.1 SUMMARY OF RISK CHARACTERIZATION BASED ON THE 95 PERCENT UCL FOR BISRA

5.3.1.1 UN-1100-6 Subunit BISRA. BEHP detected in soil at UN-1100-6 subunit is estimated to have an ICR greater than 1E-06 when using the maximum detected contaminant concentration to calculate the ICR (see table 5-1). Although chlordane did not exceed an ICR of 1E-06, it was retained for evaluation based on the 95 percent UCL value because its distribution is similar to that of BEHP in the soil. The 95 percent UCL's for BEHP and chlordane are presented in table 5-8; the associated estimated contaminant intakes for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-9; and the HQ's and ICR's for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-10.

Exposure to BEHP via the soil ingestion route is associated with an ICR of 2E-05. Dermal exposure to BEHP is estimated to have an ICR of 2E-06. For chlordane, the soil ingestion ICR is 2E-07 and the dermal exposure ICR is 2E-07. The fugitive dust pathway is associated with negligible cancer risks for both contaminants. All ICR's are the same order of magnitude as those estimated using the maximum detected contaminant concentrations. The total ICR subunit is 2E-05.

5.3.1.2 Ephemeral Pool BISRA. PCB's detected in soil at the Ephemeral Pool are estimated to have an ICR greater than 1E-06 when using the maximum detected contaminant concentration to calculate the ICR (see table 5-1). Chlordane, although it is not estimated to have an ICR greater than 1E-06 for any industrial scenario pathway, is also evaluated for this subunit.

The 95 percent UCL's for chlordane and PCB's are presented in table 5-8; the estimated contaminant intakes for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-9; and the HQ's and ICR's for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-10.

Table 5-8. 95% UCL Concentrations for Soil Contaminants Evaluated in the Baseline Industrial Scenario Risk Assessment.

Contaminants	Horn Rapids Landfill mg/kg	UN-1100-6 mg/kg	Ephemeral Pool mg/kg
Arsenic	1.4	--	--
Chromium	83	--	--
BEHP	--	18,000	--
Chlordane	--	1.6	1.9
PCBs	38	--	15

UCL = Upper Confidence Limit
-- = Not applicable

Table 5-9. Summary of Industrial Scenario Intakes Based on the 95% UCL for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill.

Contaminant	Pathway					
	Soil Ingestion (mg/kg-d)		Fugitive Dust Inhalation (mg/kg-d)		Dermal Exposure (mg/kg-d)	
	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic
UN-1100-6						
BEHP	5.3E-03	1.5E-03	--	1.6E-06	6.0E-04	1.6E-04
Chlordane	4.5E-07	1.3E-07	--	1.4E-10	5.4E-07	1.5E-07
Ephemeral Pool						
Chlordane	5.5E-07	1.6E-07	--	4.5E-10	6.5E-07	1.9E-07
PCBs	--	1.2E-06	--	3.6E-09	--	1.5E-06
Horn Rapids Landfill						
Arsenic	4.0E-07	1.1E-07	--	2.3E-10	8.0E-09	2.3E-09
Chromium	2.4E-05	--	--	4.6E-08	4.7E-07	--
PCBs	--	3.1E-06	--	2.1E-08	--	3.7E-06

*Intakes adjusted based on 30% absorption of inhaled arsenic (EPA, 1992b)
-- = Not Applicable

Table 5-10. Summary of the Baseline Industrial Scenario Risk Assessment Based on the 95% UCL for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill.

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HI[c]	ICR[b]
UN-1100-6										
BEHP	0.3	2E-05	--	2E-08	0.03	2E-06	0.3	2E-05		
Chlordane	0.008	2E-07	--	2E-10	0.009	2E-07	0.01	4E-07		
Pathway Totals	0.3	2E-05	--	2E-08	0.04	2E-06			0.3	2E-05
Ephemeral Pool										
Chlordane	0.009	2E-07	--	8E-10	0.01	2E-07	0.02	4E-07		
PCBs	--	9E-06	--	3E-08	--	1E-05	--	2E-05		
Pathway Totals	0.009	9E-06	--	3E-08	0.01	1E-05			0.02	2E-05
Horn Rapids Landfill										
Arsenic	0.001	2E-07	--	1E-08	0.00003	4E-09	0.001	2E-07		
Chromium	0.005	--	--	2E-06	0.00009	--	0.005	2E-06		
PCBs	--	2E-05	--	2E-07	--	3E-05	--	5E-05		
Pathway Totals	0.007	2E-05	--	2E-06	0.0001	3E-05			0.007	5E-05

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Hazard Index
[d]Based on 30% absorption of inhaled arsenic (EPA, 1992b)
-- = Not Applicable

The ICR's associated with the soil ingestion, fugitive dust, and dermal exposure pathways calculated using the 95 percent UCL for chlordane are 2E-07, 6E-10, and 2E-07, respectively. These are essentially the same as those estimated using the maximum detected contaminant concentrations. The ICR's for PCB's are 9E-06 and 1E-05 for the soil ingestion pathway and the dermal exposure pathway, respectively. The fugitive dust pathway is estimated to have an ICR of 3E-08 for PCB's. The total subunit ICR is 2E-05.

5.3.1.3 HRL BISRA. Three contaminants detected in the soil, arsenic, chromium, and PCB's, are estimated to have ICR's greater than 1E-06 when exposures are evaluated using the maximum detected concentrations of the contaminants. Further evaluation of these three contaminants is discussed below.

Arsenic, chromium, and PCB's, are evaluated using 95 percent UCL contaminant concentrations. As discussed in appendix IV, for arsenic, all Phase I and II sampling data are used to calculate the 95 percent UCL. The sampling for this contaminant indicates that it is evenly distributed throughout the subunit. Therefore, the 95 percent UCL represents the spatial distribution and frequency of detection for arsenic and should be representative of the potential contamination of the entire HRL. For chromium and PCB's, the site investigation and sampling data results identified some areas that appear to have generally higher concentrations of these two COPC's. Therefore, the 95 percent UCL's have been calculated based on only part of the sampling data collected at HRL. The use of the 95 percent UCL for estimating ICR's and HQ's associated with chromium and PCB's provides a hot spot evaluation.

The 95 percent UCL's for arsenic, chromium, and PCB's are presented in table 5-8; the estimated contaminant intakes for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-9; and the HQ's and ICR's for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-10.

For the soil ingestion pathway, ICR's are 2E-07 for arsenic and 2E-05 for PCB's using the 95 percent UCL. By comparison, based on the maximum detected contaminant concentration, the ICR for arsenic, and PCB are 9E-07 and 6E-05, respectively. Chromium is not considered carcinogenic by the oral route of exposure.

The ICR for chromium by the fugitive dust pathway is 2E-06 using the 95 percent UCL; whereas, for the maximum detected contaminant concentration the estimated ICR is 3E-05. The ICR's for all other COPC's are less than 1E-06 for the fugitive dust pathway.

The ICR for arsenic is less than 1E-06 for dermal exposure. PCB's are estimated to have an ICR of 3E-05 for the dermal exposure pathways, based on the 95 percent UCL, as compared to 8E-05 based on the maximum detected contaminant concentration. Chromium is not known to be carcinogenic by the dermal route. The total ICR, based on the 95 percent UCL's, is 5E-05 for HRL.

5.3.2 SUMMARY OF RISK CHARACTERIZATION BASED ON THE 95 PERCENT UCL FOR BRSRA

The results of risk characterization based on the 95 UCL are discussed in this paragraph.

As stated for BISRA, the sampling data from both the Phase I and Phase II site investigations were used to calculate the 95 percent UCL of the mean contaminant concentration at these subunits. The procedure and data used to calculate the 95 percent UCL are presented in appendix III. The 95 percent UCL provides a conservative estimate of the mean concentration. In order to provide an estimate of the potential risk due to the UN-1100-6 subunit and HRL, data from defined hot spots were used to develop a 95 percent UCL with a conservative bias. The 95 percent UCL contaminant concentration is used to estimate contaminant intakes, HQ's, and ICR's.

5.3.2.1 1100-3 BRSRA. The risk estimate for 1100-3 (9E-06) is associated with potential exposure to arsenic through the ingestion of soil, and is based on a maximum concentration of 3.4 mg/kg detected in a near surface sample. All other concentrations of arsenic were approximately one-half of that detected in the maximum, and are likely to represent typical background concentrations of arsenic in soil at this subunit. Given that the estimated risk represents a significant contribution from background arsenic in the soil and that even background concentrations may pose an ICR greater than 1E-06, no further evaluation of arsenic is made at this subunit. A characterization of risk at 1100-3 based on the 95 percent UCL is, therefore, unnecessary.

5.3.2.2 UN-1100-6 Subunit BRSRA. BEHP and chlordane detected in soil at UN-1100-6 subunit are both estimated to have ICR's greater than 1E-06 when using the maximum contaminant concentrations to calculate the ICR's (table 5-3).

The 95 percent UCL's for BEHP and chlordane are presented in table 5-11; the associated estimated contaminant intakes for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-12; and the HQ's and ICR's for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-13.

Exposure to BEHP via the soil ingestion route is associated with a HQ of 3 and an ICR of 4E-04. Dermal exposure to BEHP is estimated to have an ICR of 5E-05. For chlordane, the soil ingestion ICR is 3E-06 and the dermal exposure ICR is 4E-06. The fugitive dust pathway is associated with negligible cancer risks for both contaminants. All ICR's are within one order of magnitude of those estimated using the maximum contaminant concentrations.

The 95 percent UCL's for BEHP and chlordane in soil are also used to calculate intakes, ICR's, and HQ's, associated with the garden produce pathway. Table 5-14 presents a summary of the estimated contaminant concentrations in garden produce based on soil contaminated with BEHP and chlordane at the 95 percent UCL. A summary of the associated contaminant intakes is provided in table 5-15. A summary of the associated estimated ICR's and HQ's are provided in table 5-16. The ICR's and HQ's for BEHP and

chlordane are essentially the same whether the maximum contaminant concentration (table 5-3) or the 95 percent UCL (table 5-11) is used to evaluate potential garden produce exposures.

5.3.2.3 Ephemeral Pool BRSRA. Chlordane and PCB's detected in soil at the Ephemeral Pool are both estimated to have ICR's greater than 1E-06 when using the maximum contaminant concentrations to calculate the ICR's (table 5-3). Sampling data from both the Phase I and Phase II site investigations were used to calculate the 95 percent UCL for each contaminant at this subunit. The procedure and data used to calculate the 95 percent UCL are presented in appendix V.

The 95 percent UCL's for chlordane and PCB's are presented in table 5-11; the associated estimated contaminant intakes for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-12; and the HQ's and ICR's for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-13.

The ICR's associated with the soil ingestion, fugitive dust, and dermal exposure pathways calculated using the 95 percent UCL for chlordane are 4E-06, 1E-09, and 5E-06, respectively. These are essentially the same as those estimated using the maximum contaminant concentrations. The ICR's for PCB's are 2E-04 for both the soil ingestion pathway and the dermal exposure pathway. The fugitive dust pathway is estimated to have an ICR of 6E-08.

The 95 percent UCL's for chlordane and PCB's in soil are also used to calculate intakes, ICR's, and HQ's, associated with the garden produce pathway. Table 5-14 presents a summary of the estimated contaminant concentrations in garden produce based on soil contaminated with BEHP and chlordane at the 95 percent UCL. A summary of the contaminant intakes is provided in table 5-15. A summary of the associated estimated ICR's and associated HQ's are provided in table 5-16.

The overall garden pathway ICR, based on the 95 percent UCL is 8E-04. This is primarily attributable to the estimated ICR for PCB's, which is 7E-04.

5.3.2.4 HRL BRSRA. Contaminants detected in both the soil and the groundwater are associated with ICR's of greater than 1E-06 when exposures are evaluated using the maximum detected concentration of the contaminants (table 5-5). Further evaluation of these soil and groundwater contaminants are discussed below.

5.3.2.4.1 Soil--Four contaminants detected in soil at HRL, arsenic, beryllium, chromium, and PCB's, are evaluated using 95 percent UCL contaminant concentrations.

For beryllium, all sampling data are used to calculate the 95 percent UCL. The sampling for this contaminant indicates that the contaminant is evenly distributed throughout the subunit. Therefore, the 95 percent UCL represents the spatial distribution and frequency of detection for this contaminant and should be representative of the potential contamination of the entire HRL.

Table 5-11. 95% UCL Concentrations for Soil Contaminants Evaluated in the Baseline Residential Scenario Risk Assessment.

Contaminants	Horn Rapids Landfill mg/kg	UN-1100-6 mg/kg	Ephemeral Pool mg/kg
Arsenic	1.4	--	--
Beryllium	0.5	--	--
Chromium	83	--	--
BEHP	--	18,000	--
Chlordane	--	1.6	1.9
PCBs	38	--	15

UCL = Upper Confidence Limit
-- Indicates not applicable

Table 5-12. Summary of Residential Scenario Intakes Based on the 95% UCL for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill.

Contaminant	Pathway					
	Soil Ingestion (mg/kg-d)		Fugitive Dust Inhalation (mg/kg-d)		Dermal Exposure (mg/kg-d)	
	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic
UN-1100-6						
BEHP	6.7E-02	2.9E-02	--[c]	3.5E-06	8.0E-03	3.4E-03
Chlordane	5.8E-06	2.5E-06	--[c]	3.0E-10	7.5E-06	3.2E-06
Ephemeral Pool						
Chlordane	7.0E-06	3.0E-06	--[c]	9.6E-10	9.0E-06	3.9E-06
PCBs	--	2.4E-05	--[c]	7.7E-09	--[c]	3.1E-05
Horn Rapids Landfill						
Arsenic	5.1E-06	2.2E-06	--[c]	5.1E-10	1.1E-06	4.8E-06
Beryllium	2.0E-06	8.6E-07	--[c]	6.6E-10	4.4E-06	1.9E-06
Chromium	3.0E-04	--[b]	--[c]	9.9E-06	6.6E-06	--[b]
PCBs	--[c]	6.0E-05	--[c]	4.5E-06	--[c]	7.8E-05

[a]Intakes adjusted based on 30% absorption of inhaled arsenic (EPA, 1992b)
[b]Not considered carcinogenic by this route of exposure or pathway
[c]RfD not available to evalute intake for this pathway
[d]SF not available to evaluate intake for this pathway
UCL = Upper Confidence Limit
-- Indicates not Applicable

Table 5-13. Summary of the Baseline Residential Scenario Risk Assessment Based on the 95% UCL for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill.

Contaminant	Pathway						Contaminant Totals		Subunit Totals	
	Soil Ingestion		Fugitive Dust Inhalation		Dermal Exposure					
	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HQ[a]	ICR[b]	HI[c]	ICR[b]
UN-1100-6										
BEHP	3	4E-04	--	5E-08	0.4	5E-05	3.4	4E-04		
Chlordane	0.1	3E-06	--	4E-10	0.1	4E-06	0.2	7E-06		
Pathway Total	3	4E-04	--	5E-08	0.5	5E-05			4	4E-04
Ephemeral Pool										
Chlordane	0.1	4E-06	--	1E-09	0.2	5E-06	0.3	9E-06		
PCBs	--	2E-04	--	6E-08	--	2E-04	--	4E-04		
Pathway Totals	0.1	2E-04	--	6E-08	0.2	2E-04			0.3	4E-04
Horn Rapids Landfill										
Arsenic	0.02	4E-06	--[e]	3E-08[d]	0.0004	9E-08	0.02	4E-06		
Beryllium	0.0004	4E-06	--[e]	6E-09	0.000009	8E-08	0.0004	4E-06		
Chromium	0.06	--[f]	--[e]	4E-06	0.001	--[f]	0.07	4E-06		
PCBs	--[e]	5E-04	--[e]	3E-07	--[e]	6E-04	--[e]	1E-03		
Pathway Total	0.08	5E-04	--[e]	4E-06	0.001	6E-04			0.08	1E-03

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Hazard Index
[d]Based on 30% absorption of inhaled arsenic (EPA, 1992b)
[e]RfD not available to evaluate this pathway
[f]Not considered carcinogenic by this route of exposure
[g]SF not available to evaluate this pathway
UCL = Upper Confidence Limit
-- Indicates not Applicable

Table 5-14. Summary of Contaminant Concentrations for the Garden Pathway at UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill Based on the 95% UCL.

	Leafy (lettuce) (mg/kg)	Root (carrots) (mg/kg)	Garden Fruits (tomatoes) (mg/kg)	Potatoes (mg/kg)
UN-1100-6				
BEHP	7E+03	6.6E+03	3.7E+02	3.7E+02
Chlordane	3.2E-02	3.2E+00	3.3E-01	4.8E-01
Ephemeral Pool				
Chlordane	3.8E-02	3.8E+00	4.0E-01	5.7E-01
PCB	5.7E+00	5.4E+00	3.0E-01	3.0E-01
Horn Rapids Landfill				
Arsenic	5.6E-02	2.8E-02	2.8E-03	8.4E-04
Beryllium	2.4E-01	1.4E-01	2.3E-02	3.3E-02
Chromium	1.7E+01	2.2E+01	3.4E+00	5E+00
PCB	1.4E+01	1.4E+01	7.6E-01	7.6E-01
UCL = Upper Confidence Limit				

Table 5-15. Summary of Contaminant Intakes for Homegrown Vegetables in the Garden Pathway at UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill Based on the 95% UCL.

	Leafy (lettuce)[a] (mg/kg-d)		Root (carrots)[b] (mg/kg-d)		Garden Fruits (tomatoes)[c] (mg/kg-d)		Potatoes[d] (mg/kg-d)		Total Contaminant Intake (mg/kg-d)	
	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic	Non-Carcinogenic	Carcinogenic
UN-1100-6										
BEHP	1.1E-01	4.5E+02	8.1E-02	3.4E-02	1.1E-02	4.8E-03	4.7E-02	2.0E-02	2.5E-01	1.0E-01
Chlordane	5E-07	2.1E-07	3.9E-05	1.7E-05	1.0E-05	4.3E-06	6.1E-05	2.6E-05	1.1E-04	4.7E-05
Ephemeral Pool										
Chlordane	5.8E-07	2.5E-07	4.7E-05	2.0E-05	1.2E-05	5.2E-06	7.3E-05	3.1E-05	1.3E-04	5.6E-05
PCBs	--[f]	3.7E-05	--[f]	2.8E-05	--[f]	3.9E-06	--[f]	1.6E-05	--[f]	8.5E-05
Horn Rapids Landfill										
Arsenic	8.6E-07	--[e]	3.4E-07	--[e]	8.6E-08	--[e]	1.1E-07	--[e]	1.4E-06	--[e]
Beryllium	3.7E-06	1.6E-06	1.7E-06	7.3E-07	7.1E-07	3E-07	4.2E-06	1.8E-06	1.0E-05	4.4E-06
Chromium	2.6E-04	--[e]	2.7E-04	--[e]	1.0E-04	--[e]	6.4E-04	--[e]	1.3E-03	--[e]
PCBs	--[f]	9.1E-05	--[f]	7.3E-05	--[f]	9.9E-06	--[f]	4.1E-05	--[f]	2.2E-04

[a]Assumes intake of 0.88 g/d dry weight (EPA, 1986a)
[b]Assumes intake of 1.1 g/d dry weight (EPA, 1986a)
[c]Assumes intake of 2.2 g/d dry weight (EPA, 1986a)
[d]Assumes intake of 9.1 g/d dry weight (EPA, 1986a)
[e]Not considered carcinogenic by this route of exposure or pathway
[f]RfD not available to evaluate intake for this pathway
[g]SF not available to evaluate
UCL = Upper Condence Limit
-- Indicates not applicable

Table 5-16. Summary of Baseline Residential Scenario Risk Assessment for the Garden Pathway at UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill Based on the 95% UCL.

Contaminant	Pathway	
	Garden	
	HQ[b]	ICR[a]
UN-1100-6		
BEHP	13	2E-03
Chlordane	1.8	6E-05
Total Pathway ICR	--	2E-03
Total Pathway HI[c]	15	--
Ephemeral Pool		
Chlordane	2.2	7E-05
PCBs	--[e]	7E-04
Total Pathway ICR	--	8E-04
Total Pathway HI[c]	2.2	--
Horn Rapids Landfill		
Arsenic	0.005	--[d]
Beryllium	0.002	2E-05
Chromium	0.3	--[d]
PCBs	--[e]	2E-03
Total Pathway ICR	--	2E-03
Total Pathway HI[c]	0.3	--

[a]Lifetime Incremental Cancer Risk
[b]Hazard Quotient
[c]Hazard Index
[d]Not considered carcinogenic by this route of exposure
[e]RfD not available to evaluate this pathway
[f]SF not available to evaluate this pathway
UCL = Upper Confidence Limit
-- Indicates not applicable

The 95 percent UCL's for arsenic, beryllium, chromium, and PCB's are presented in table 5-11; the estimated associated contaminant intakes for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-12; and the associated HQ's and ICR's for the soil ingestion, fugitive dust inhalation, and dermal exposure pathways are presented in table 5-13.

For the soil ingestion pathway, the ICR for beryllium (4E-06) exceeds 1E-06 (Table 5-13). By comparison, based on the maximum detected contaminant concentration, the ICR for beryllium is (9E-06) (table 5-1). The ICR for beryllium is less than 1E-06 for the fugitive dust pathway.

The 95 percent UCL's in soil are also used to calculate intakes, ICR's, and HQ's, associated with the garden produce pathway. Table 5-14 presents a summary of the estimated contaminant concentrations in garden produce based on soil contaminated with arsenic, beryllium, chromium, and PCB's at the 95 percent UCL concentration. A summary of the associated contaminant intakes is provided in table 5-15. A summary of the associated estimated ICR's and HQ's are provided in table 5-16.

For the garden produce pathway, beryllium is estimated to have an ICR of 2E-05 and PCB's are estimated to have an ICR of 2E-03.

5.3.2.4.2 Groundwater--Two contaminants detected in the groundwater in the vicinity of HRL are trichloroethene and nitrate. In addition to these contaminants, gross alpha and gross beta activity were detected at levels that exceed the drinking water criteria during some sampling rounds. Therefore, additional evaluation of the trichloroethene, nitrate, and elevated radioactivity based on the 95 percent UCL is presented.

Data from a select group of wells has been used to calculate the 95 percent UCL's for the contaminants. Further information on the wells selected and the data are provided in appendix IV. For trichloroethene, data from wells that have trichloroethene consistently detected above the MCL of 5 μg/L are used for the statistical calculation of the 95 percent UCL. For nitrate, wells with data exceeding the nitrate MCL of 10 mg/L are used for the calculations. The selection of these wells incorporates a conservative bias in the calculation of the ICR's and HQ's. The frequency of detection and the spatial distribution used to provide a 95 percent UCL is representative of the groundwater quality within the contaminant plume.

The 95 percent UCL's for trichloroethene and nitrate in groundwater are presented in table 5-17. A summary of the associated estimated intakes for these contaminants based on the 95 percent UCL's is provided in table 5-18. The associated estimated ICR's and HQ's are presented in table 5-19. The HQ estimated for potential ingestion of nitrate at the 95 percent UCL is 0.8, as compared to a HQ of 1 based on the maximum detected concentration (table 5-3). Trichloroethene is estimated to have an ICR of 1E-05 for the ingestion pathway and an ICR of 2E-05 for the inhalation of trichloroethene during groundwater use in a residence. The ICR's are essentially the same as those estimated for exposures to the maximum detected concentration detected in the groundwater where the ICR for ingestion is also 1E-05 and for inhalation is 3E-05 (table 5-3).

Table 5-17. 95% UCL Concentrations for Trichloroethene and Nitrate in Groundwater at the Horn Rapids Landfill.

Contaminant	Concentration (mg/L)
Nitrate	45
Trichloroethene	0.075
UCL = Upper Confidence Level	

Table 5-18. Summary of Residential Scenario Intakes Based on the 95% UCL Concentrations for the Groundwater Pathway at the Horn Rapids Landfill.

Contaminant	Pathway			
	Ingestion (mg/kg-d)		Volatile Inhalation (mg/kg-d)	
	Noncarcinogenic	Carcinogenic	Noncarcinogenic	Carcinogenic
Nitrate	1.23E+00	--[a]	--[b]	--[a,b]
Trichloroethene	--[c]	8.8E-04	--[c]	3.3E-03

[a]Not considered to be a carcinogen
[b]Not a volatile contaminant
[c]RfD not available to evaluate intake for this pathway
UCL = Upper Confidence Level
-- Indicates not applicable

Table 5-19. Summary of Baseline Residential Scenario Risk Assessment Based on the 95% UCL Concentrations for the Groundwater Pathway at the Horn Rapids Landfill.

Contaminant	Pathway			
	Groundwater Ingestion		Groundwater Inhalation	
	HQ[a]	ICR[b]	HQ[a]	ICR[b]
Nitrate	0.8	--[c]	--[d]	--[c,d]
Trichloroethene	--[e]	1E-05	--[e]	2E-05

[a]Hazard Quotient
[b]Lifetime Incremental Cancer Risk
[c]Not considered to be a carcinogen
[d]Not a volatile contaminant
[e]RfD not available to evaluate this pathway
UCL = Upper Confidence Level
-- Indicates not applicable

Gross alpha and gross beta activity has also been detected in the groundwater in the vicinity of HRL. The 95 percent UCL's for both gross alpha and gross beta are 5 pico Curie/liter (pCi/L) and 65 pCi/L, respectively. The current MCL for gross alpha activity (excluding radon and uranium) is 15 pCi/L. In addition, since the gross alpha measurement does not exceed 5 pCi/L, compliance with the MCL's for Ra-226 and Ra-228 may be assumed without further analysis. An MCL for gross beta activity has not been developed. However, compliance with individual MCL's for beta emitters may be assumed without further analysis if the average annual concentration of gross beta activity is less than 50 pCi/L. Since the gross beta activity exceeds this concentration, an analysis of the sample to identify the major radioactive constituents present is required and was performed (see below).

Although gross alpha and gross beta measurements can provide an indication of radioactive contamination, such values are of limited usefulness in risk assessment. This is because slope factors are radionuclide-specific, and associated risks cannot be calculated from gross alpha and gross beta measurements when the relative proportions of various sediments are known.

More specific analysis of the potential beta-contributing radionuclides was conducted. Technetium-99 appears to account for most, if not all, of this beta activity, and no other significant contributors to the total beta activity have been detected (Prentice *et al.*, 1992). Other analyses performed were tritium, Sr-90, liquid scintillation, and gamma spectrometry. Tc-99 is a fission product released to the environment mainly from recycling of nuclear fuels, and is very persistent with a half-life of 2.1E+05 yr. It has a relatively small ingestion slope factor (1.3E-12/pCi), indicating that this radionuclide poses a relatively small internal hazard. This is also indicated by the high proposed MCL for Tc-99 (3,800 pCi/L). The average Tc-99 concentration measured in the plume was 120 pCi/L. Under a residential scenario, the lifetime incremental cancer risk associated with this concentration in drinking water is approximately 3E-06.

A summary of the pathway and subunit ICR's and HQ's based on the 95 percent UCL is presented for UN-1100-6 subunit, the Ephemeral Pool, and HRL in table 5-20.

The subunit ICR for UN-1100-6 subunit is 2E-03 because of the potential risks associated with garden produce. At the Ephemeral Pool, the subunit ICR is 1E-03. The garden produce pathway contributes a pathway ICR of 8E-04. The soil ingestion and dermal exposure pathways are both estimated to have ICR's of 2E-04. The HRL subunit ICR is 3E-03. Again, the garden produce pathway (ICR = 2E-03) contributes most of the risk.

5.4 RISK CHARACTERIZATION UNCERTAINTY ANALYSIS

A human risk characterization examines the sources of the contaminant, its dispersion in the environment and resulting exposure to humans, and the toxicological effects of such exposure. The risks, both carcinogenic and noncarcinogenic, presented in this risk assessment are conditional estimates given multiple assumptions about exposures, toxicities,

and other variables. This discussion focuses on the uncertainties surrounding the projected risks and hazards due to uncertainty in these variables.

5.4.1 Uncertainty Associated with the Identification of COPC's

The soil sampling conducted under the Phase I and Phase II RI's provides confidence that the COPC's at the 1100-EM-1 Operable Unit have been identified. Phase II sampling confirmed sampling data from the earlier remedial investigation activities except as noted below. Additional COPC's have been identified and evaluated in the BISRA because of the more conservative risk-based screening procedure used relative to DOE/RL-90-18 (*e.g.*, ICR = 1E-07 and HQ = 0.1), the availability of new toxicity information (*e.g.*, regarding beryllium), and additional sampling data and maximum concentrations (*e.g.*, regarding PCB's). However, overall results are consistent with the results of the Phase I RI Report.

Two parameters were detected in the Phase II soil sampling at HRL that require additional consideration for the residential risk assessment. Dieldrin has been detected at a maximum concentration of 1.2 mg/kg. Recent data validation has revealed that concentrations reported for Dieldrin are "qualified," which indicates that the case narrative from the lab should be consulted. Upon review, the analysts's opinion is that Dieldrin is actually a part of the Arochlor pattern. Therefore, Dieldrin has not been evaluated as a COPC.

Alpha chlordane has also been detected at a maximum concentration of 0.78 mg/kg in the Phase II sampling at HRL, but is also qualified. It has also been detected at 0.41 mg/kg without any qualifiers nearby. Although not evaluated as a contaminant of concern, chlordane, at either of these concentrations, would not be associated with a risk greater than 1E-06 based on the industrial scenario evaluated in the BISRA. By comparison, the risks for chlordane at UN-1100-6 subunit (detected at about 1.9 mg/kg) are associated with a contaminant-specific ICR of 4E-07 (summary of chlordane ICR's presented in table 5-1 for UN-1100-6) for the soil ingestion, fugitive dust, and dermal exposure pathways. This would correspond approximately to an ICR of 2E-07 for a concentration of 0.78 mg/kg or 9E-08 for a concentration of 0.41 mg/kg. Consequently, there is uncertainty in the contribution of chlordane to the overall risk estimate for HRL, but it appears that the contribution to the overall subunit risk in the BISRA, would be low.

Although not evaluated as a contaminant of concern, chlordane at either of the above concentrations could be associated with a risk greater than 1E-06 based on the residential scenario evaluated in the BRSRA. As a comparison, the risks for chlordane at UN-1100-6 subunit (detected at 1.9 mg/kg) are associated with a contaminant-specific ICR of 8E-05 (summary of chlordane ICR's presented in tables 5-1 and 5-2 for UN-1100-6 subunit) for the soil ingestion, fugitive dust, dermal exposure, and garden pathways. This would correspond approximately to an ICR of 3E-05 for a concentration of 0.78 mg/kg or 2E-05 for a concentration of 0.41 mg/kg. Consequently, there is uncertainty in the contribution of chlordane to the overall risk in the BRSRA estimate for HRL.

Table 5-20. Summary of the Baseline Residential Scenario Risk Assessment for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill Based on the 95% UCL.

Subunit	Pathway	Pathway Totals		Subunit Totals	
		HI[a]	ICR[b]	HI[a]	ICR[b]
UN-1100-6	Soil Ingestion	3.0	4E-04		
	Fugitive Dust Inhalation	--	5E-08		
	Dermal Exposure	0.5	5E-05		
	Garden Produce	15	2E-03		
				18	2E-03
Ephemeral Pool	Soil Ingestion	0.1	2E-04		
	Fugitive Dust Inhalation	--	8E-08		
	Dermal Exposure	0.2	2E-04		
	Garden Produce	2.2	8E-04		
				2.5	1E-03
Horn Rapids Landfill	Soil Ingestion	0.08	5E-04		
	Fugitive Dust Inhalation	--	4E-06		
	Dermal Exposure	0.001	8E-04		
	Garden Produce	0.3	2E-03		
	Groundwater Ingestion	0.8	1E-05		
	Inhalation of Volatiles from Groundwater	--	2E-05		
				1.2	3E-03

[a]Hazard Index
[b]Lifetime Incremental Cancer Risk
-- Indicates not applicable

Beryllium is a COPC that has been evaluated in the BRSRA for HRL because of new toxicity information that was not available when the Phase I RI was prepared.

5.4.2 Uncertainty Associated with the Exposure Assessment

The exposure assessment is based on a large number of assumptions regarding the physical setting of the 1100-EM-1 Operable Unit, and the exposure conditions of the receptor population. For the purpose of the BISRA, a conservative assumption is made that the COPC's being evaluated are readily accessible for worker contact via ingestion, inhalation and dermal exposure pathways. Actual site conditions, however, may substantially limit or preclude such exposures. In most cases, the maximum concentrations detected are not uniformly distributed in the soil and may be several feet below the surface. At subunit 1100-4, the contamination is located inside a building under a cement floor. For the purpose of the BRSRA, a conservative assumption is made that the COPC's being evaluated are readily accessible for receptor contact via ingestion, inhalation, dermal, and garden produce pathways. Actual site conditions, however, may substantially limit or preclude such exposures. For example, residential use of the area in the foreseeable future is unlikely.

The fugitive dust inhalation pathway utilizes a number of assumptions, including potential for soil erodability, soil grain-size distribution, length of each operable subunit relative to the prevailing wind, and other climatic factors. Conservative parameter values are chosen when site-specific information is not available. In general, use of FDM should provide appropriate, but conservative, estimates of fugitive dust because the model incorporates actual site meteorological data.

Uncertainty in the fugitive dust inhalation pathway is also present because of the lack of information relating the concentration of a contaminant with the particle size fraction. Concentrations may be greater in the fine fractions because of the greater surface area of these particles, resulting in selective partitioning of contaminants to the fine fractions.

Exposure parameters (*i.e.*, body weight, averaging time, contact rate, exposure frequency, and exposure duration) are generally conservative default parameters that represent reasonable maximum values as defined by EPA (EPA-10, 1991) and in the HSBRAM (DOE/RL-91-45), but may not reflect actual exposure conditions. For example, the soil ingestion exposure pathway uses the assumption that a resident or worker is present and ingesting dirt from the same site 350 d/yr for 30 years (residential scenario) or 146 d/yr for 20 years (industrial scenario).

Another example of conservative exposure parameter assumptions is found in the fugitive dust pathway. It is assumed that for the industrial scenario that workers are outside during the entire working lifetime and inhaling the estimated fugitive dust concentrations presented in table 3-1. In reality, current 1100 Area employees are inside various facilities and not working outside for 250 d/yr for 20 yr. Climatic conditions at the Hanford site would also limit such conservative assumptions from actually occurring. The assumption for residential scenario is that residents are outside during the entire 30 years and inhaling the estimated airborne concentrations presented in table 3-1. In reality, residents would be inside

homes, away at school or jobs, and not working or playing outside for a large portion of the 350 d/yr for 30 yr.

The choice of intake parameters for all exposure pathways is governed by the specific land use evaluated. Any land use change that would increase exposures by workers or indicate a different receptor population would result in a need to reevaluate the risks presented here.

The inhalation of volatile contaminants present in soil or soil gas has not been quantitatively evaluated in the BRSRA. Tetrachloroethene has been detected at very low concentrations in soil at 1100-2 and HRL. Tetrachloroethene, trichloroethene, and 1,1,1-trichloroethane have been detected in soil gas at HRL. Although inhalation of these volatile COPC's could occur if residents lived on the landfill, the low concentrations detected suggest that this would not result in unacceptable exposures. This is further supported by modelling performed in the Phase I RI report and the results of additional soil gas surveys during the Phase II RI. However, the lack of a quantified evaluation may result in an underestimate of the total site risk.

The garden produce pathway utilizes conservative uptake factors to estimate concentrations of contaminants in the plants. Actual subunit soil conditions could affect the uptake. In addition, the assumption that the garden is located at the site of the maximum contaminant concentration or the 95 percent UCL concentration is conservative since these areas usually represent only a portion of the entire subunit. The exceptions are UN-1100-6 subunit and the Ephemeral Pool.

The choice of intake parameters for all exposure pathways is governed by the specific land use evaluated. This assessment considers only an onsite residential scenario which assumes that there will be major changes in current land use at the Operable Unit. This seems improbable based on current land use, zoning, and restrictions related to the Hanford Site.

The spatial distribution of chromium from the Phase I RI suggests that high concentrations are confined to a small area of HRL and are not uniformly distributed in the soil. Estimations based on maximum concentrations and 95 percent UCL in general would overestimate actual risks, where use of data collected over the entire landfill may underestimate risks from exposure to hot spots. Natural background conditions are not considered in the evaluation of the estimated ICR's for any of the COPC's. In some cases, for example arsenic, natural background concentrations may be associated with risks that would be potentially unacceptable at a remediated NPL site.

In the control-screening process, parameters detected below project-specific background (*i.e.*, UTL) were not considered background. This process was approved for use according to the version of HSBRAM (DOE/RL-91-45,1992) followed for this risk assessment. The HSBRAM is currently undergoing revision, and the final form may not recommend control-screening in this manner for organic parameters. To determine if the organic parameters below UTL's would contribute significantly to the risk, EPA requested that maximum concentrations of these parameters be compared to risk-based concentrations

for soil ingestion. This was presented at the October 1992 Hanford site unit manager's meeting. Risk calculations performed for contaminants below background showed that these contaminants would not contribute to the overall risk.

Absorption factors of contaminants from soil have been derived to evaluate the dermal absorption pathway. Limited data are available on the absorption of chemicals from a soil matrix. Therefore, the assessment of risks may be an overestimation or an underestimation of the actual risk.

5.4.3 Uncertainty Associated with the Toxicity Assessment

Uncertainty is also associated with the toxicity values and toxicity information available to assess potential adverse effects. This uncertainty in the information and the lack of specific toxicity values for some COPC's contribute to uncertainty in the toxicity assessment.

5.3.4.1 Uncertainty in Toxicity Values and Information. An understanding of the degree of uncertainty associated with toxicity values is an important part of interpreting and using those values. A high degree of uncertainty in the information used to derive a toxicity value contributes to less confidence in the assessment of risk associated with exposure to a substance.

The RfD's and SF's have multiple conservative calculations built into them that can contribute to overestimation of actual risk (*i.e.*, factors of 10 for up to four different levels of uncertainty for RfD's, and the use of a 95 percent upperbound confidence estimate derived from the linearized multi-stage carcinogenic model for SF's). For example, table 4-1 indicates that an uncertainty factor of 1,000 is used to calculate the RfD's for chlordane and tetrachloroethene. Table 4-2 shows that, while beryllium, BEHP, chlordane, and PCB's are evaluated as human carcinogens, the available information indicates that there is inadequate evidence of carcinogenicity in humans. The extrapolation of data from high-dose animal studies to low-dose environmental human exposures may overestimate the risk in the human population because of metabolic differences, repair mechanisms, or different susceptibilities.

An underestimation of systemic toxicity could be associated with the inhalation pathway because only one COPC, barium, has a published inhalation RfD. The RfD for barium is an interim number based on short term reproductive studies in rats and is under review.

5.4.3.2 Uncertainty in the Toxicity Assessment. Uncertainty is also present in the overall toxicity assessment for several reasons. First, substances have been evaluated qualitatively when there is a lack of toxicity values. Second, route specific toxicity values have been extrapolated from one route to another (*e.g.*, oral to dermal). Additionally, surrogate values are used and potential synergistic or antagonistic interactions of substances have not been evaluated.

Conservative assumptions are provided regarding the species of the contaminant present. For example, all chromium is assumed to be chromium(VI) which is carcinogenic.

Toxicity values are not available for several contaminants detected at the subunits (*e.g.*, lead can have significant toxic effects. In addition, the form of lead present may also affect the toxicity because some compounds are more bioavailable than others. Because many of the effects for lead toxicity are apparently without a threshold, the EPA does not provided numerical toxicity values. Lead has been evaluated using the UBK model, which is based on conservative assumptions of the form of lead that may be present, and consequently, assumes a form of lead that is very bioavailable. Lead was not retained as a COPC at any of the subunits because the maximum concentrations detected are within the range of the recommended soil cleanup guideline of 500 to 1,000 mg/kg (EPA, 1989b). However, lead is retained as a contaminant of interest at HRL because it exceeds the soil concentration (500 mg/kg) associated with increased blood levels in children. Children are a sensitive subpopulation for lead exposures. Lead at the concentrations detected is unlikely to pose an unacceptable hazard to workers under the industrial scenario. However, if the expected land use at the 1100 Area were to change, it may require more extensive evaluation might be warranted.

Some contaminants, such as PCB's, only have toxicity values for carcinogenic effects (*i.e.*, SF's), but do not have toxicity values for noncarcinogenic effects (*i.e.*, RfD's). These contaminants are known to produce systemic toxic effects in addition to cancer. Without an RfD, quantitative evaluation of these other effects is limited. However, the potential to cause cancer is usually the effect of most concern and is usually the effect that drives risks at most sites. As indicated, surrogates are used to evaluate COPC's when numerical toxicity values are not available. For all COPC's, the level of confidence that key effects have been evaluated is high.

The uncertainty surrounding dermal exposures and absorption from dermal exposure is another significant source of uncertainty. The lack of toxicity information to adequately determine RfD's and SF's for dermal exposures forces extrapolation from oral toxicity values, and increases the conservative bias associated with these calculations. This conservatism is reflected in the significant estimated risks associated with this pathway for some compounds, most notably PCB's. Conversely, the assumption that dermal toxicity values are the same as oral toxicity values could underestimate the risk for contaminants that are poorly absorbed in the gastrointestinal tract, but might well be absorbed dermally.

This page left intentionally blank.

6.0 SUMMARY OF THE BISRA AND THE BRSRA

The BISRA and the BRSRA have been conducted as recommended in the HSBRAM (DOE/RL-91-45), and the direction of EPA [Einan, 1991 (see appendix I)], respectively. The BISRA was prepared prior to completion of the Phase II RI for the 1100-EM-1 Operable Unit. The data from the Phase I RI, and additional sampling data from Phase II sampling at the Ephemeral Pool and HRL, were included in the BISRA. Contaminants have been determined by comparison of maximum detected concentrations of parameters to the UTL for that parameter. A BRSRA for an onsite residential scenario at each of five 1100-EM-1 operable subunits, as defined in letters [Einan, 1991, and Einan, 1992 (see appendix I)]. The scope of the BRSRA defined by these letters included evaluation of specific COPC and specific exposure pathways. The COPC derived from the comparisons for both the BISRA and the BRSRA are presented in table 2-1. The BISRA and the BRSRA initially were conducted independently and later were combined for the purposes of the RI/FS.

The maximum concentrations of COPC detected at each subunit are evaluated at each of the designated subunits. As discussed in chapter 5, paragraph 5.4, conservative assumptions have been made with respect to the species of the contaminant present. For three subunits, UN-1100-6 subunit, the Ephemeral Pool, and HRL, soil contaminants that are estimated to have an ICR greater than 1E-06, based on the maximum detected contaminant concentration, are also evaluated using a 95-percent UCL concentration. An overall pathway and subunit comparison based on the specific COPC that exceeded 1E-06 using the maximum detected contaminant concentration and the 95-percent UCL is provided in table 5.3.

As discussed in appendix IV, the 95-percent UCL for COPC at the Ephemeral Pool are based on all data for that subunit. The 95-percent UCL is used to evaluate chlordane and BEHP at UN-1100-6 subunit. At HRL, the 95-percent UCL for arsenic is based on data collected throughout the landfill. For chromium and PCB's, the 95-percent UCL concentrations reflect data selected to evaluate the areas of maximum contamination (*i.e.*, hot spots). Therefore, the 95-percent UCL's are not directly comparable between contaminants. Consequently, although a quantitative comparison is presented, the results should be carefully interpreted and emphasis should be placed on the qualitative nature of the results.

6.1 BISRA

Currently, no workers are assigned to work at any of the subunits on an ongoing basis. For purposes of the BISRA, it is assumed, based on current land use and zoning in the 1100 Area, that industrial workers are likely potential receptors at the subunits. The current and future receptor population that has been evaluated is onsite industrial workers who are assumed to work full time at only one subunit where they could potentially be exposed to contaminants from that subunit. The BISRA also assumes that personnel are assigned to the 1100 Area for purposes other than remediation.

The exposure pathways are those indicated for the industrial scenario defined in the HSBRAM (DOE/RL-91-45). The BISRA evaluates only pathways associated with exposure to soils (*i.e.*, soil ingestion, dermal exposure to soil, and fugitive dust inhalation). Potential exposures associated with groundwater and surface water are not evaluated in the BISRA. As discussed in chapter 3, paragraph 3.2, neither groundwater use nor direct use of surface water occurs because of the availability of city of Richland water services.

The air inhalation pathway assumes exposure to contaminated dust directly at each subunit. The EPA FDM is used to estimate concentrations of airborne particulates at a site based on conservative estimations of soil and climatic conditions. Chromium present in the soil at HRL is the only contaminant that may be associated with risks greater than 1E-06. However, all chromium is assumed to be chromium(VI) which is a conservative assumption as discussed in chapter 5, paragraph 5.4

Given the above considerations, the BISRA identifies and evaluates the contaminants that are most likely to pose a potential human health risk. A review of the results presented in tables 5-1 through 5-5, and summarized in table 6-1, is discussed below for each subunit.

6.3 BRSRA

The BRSRA has been conducted as recommended in RAGS (EPA, 1989a) and by (EPA-10, 1991). Currently there is no residential use of any of the subunits. The 1100-EM-1 Operable Unit is used for industrial purposes and is surrounded by land zoned by the city of Richland for industrial or commercial uses. For purposes of the BRSRA, as directed by EPA, it is assumed that residents are living at 1100-2, 1100-3, UN-1100-6 subunit, HRL, and the Ephemeral Pool. It is assumed that such individuals live only at one subunit where they could potentially be exposed to contaminants from that subunit.

As defined by EPA (Einan 1991) and a follow up letter of clarification [Einan, 1992 (see appendix I)], the exposure pathways are focused on contaminated soil. The pathways include the ingestion of soil, dermal contact with soil, ingestion of garden produce, and inhalation of particulates (*i.e.*, fugitive dust). Other pathways evaluated as discussed below, include ingestion of groundwater and recreational exposures through swimming in the Columbia River or eating fish from the Columbia River.

The dermal contact with soil pathway utilizes absorption factors to estimate the absorption of contaminants from soil through the skin of the receptor. The garden produce pathway, similarly, uses plant uptake factors to estimate the transport of contaminants from the soil to the plant. Discussions of the conservative assumptions for these pathways are provided in chapters 3 and 5, paragraphs 3.3.2 and 5.4, respectively. Both pathways are associated with relatively high ICR's (see tables 5-1, 5-2, 5-15, and 6-2). These pathways have a great deal of uncertainty associated with them because the transport of soil-bound contaminants across skin and the uptake of contaminants by plants are not well understood.

Table 6-1. Comparison of the Baseline Industrial Incremental Cancer Risk Assessment Results using the Maximum Contaminant Concentrations and 95% UCL for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill.

Subunit	Pathway	95% UCL Pathway Totals	Maximum Concentration Pathway Totals	95% UCL Subunit Totals	Maximum Concentration Subunit Totals
		ICR	ICR	ICR	ICR
UN-1100-6	Soil Ingestion	2E-05	3E-05		
	Fugitive Dust Inhalation	2E-08	3E-08		
	Dermal Exposure	2E-06	3E-06		
				2E-05	3E-05
Ephemeral Pool	Soil Ingestion	9E-06	3E-05		
	Fugitive Dust Inhalation	3E-08	8E-08		
	Dermal Exposure	1E-05	3E-05		
				2E-05	6E-05
Horn Rapids Landfill	Soil Ingestion	2E-05	6E-05		
	Fugitive Dust Inhalation	2E-08	3E-05		
	Dermal Exposure	3E-05	8E-05		
				5E-05	2E-04

Table 6-2. Comparison of the Baseline Residential Scenario Risk Assessment Results using the Maximum Contaminant Concentrations and 95% UCL for UN-1100-6, the Ephemeral Pool, and the Horn Rapids Landfill.

Subunit	Pathway	95% UCL Pathway Totals		Maximum Concentration Pathway Totals		95% UCL Subunit Totals		Maximum Concentration Subunit Totals	
		HI[a]	ICR[b]	HI[a]	ICR[b]	HI[a]	ICR[b]	HI[a]	ICR[b]
UN-1100-6	Soil Ingestion	3.0	4E-04	4.7	6E-04				
	Fugitive Dust Inhalation	--	5E-08	--	7E-08				
	Dermal Exposure	0.5	5E-05	0.7	8E-05				
	Garden Produce	15	2E-03	18	2E-03				
						18	2E-03	23	3E-03
Ephemeral Pool	Soil Ingestion	0.1	2E-04	0.2	5E-04				
	Fugitive Dust Inhalation	--	6E-08	--	2E-07				
	Dermal Exposure	0.2	2E-04	0.2	7E-04				
	Garden Produce	2.2	8E-04	3.2	2E-03				
						2.5	1E-03	3.8	3E-03
Horn Rapids Landfill	Soil Ingestion	0.08	5E-04	1	1E-03				
	Fugitive Dust Inhalation	--	4E-06	--	8E-05				
	Dermal Exposure	0.001	6E-04	0.02	2E-03				
	Garden Produce	0.3	2E-03	3.6	4E-03				
	Groundwater Ingestion	0.8	1E-05	1	1E-05				
	Inhalation of Volatiles from Groundwater	--	2E-05	--	3E-05				
						1.2	3E-03	5.6	7E-03

[a]Hazard Index
[b]Lifetime Incremental Cancer Risk
UCL Upper Confidence Limit
-- indicates not applicable

Consistent with the BISRA, the air inhalation pathway assumes exposure to contaminated dust directly at each subunit. The EPA FDM is used to estimate concentrations of airborne particulates at a site based on conservative estimations of soil and climatic conditions. Chromium present in the soil at HRL is the only contaminant that may be associated with risks greater than 1E-06. However, all chromium is assumed to be chromium(VI) which is a very conservative assumption as discussed in chapter 5, paragraph 5.4.

The EPA also directed that potential exposures through pathways associated with use of groundwater at HRL should be evaluated in the BRSRA. The evaluation of nitrate in the groundwater indicates a HQ of 0.8, if wells with nitrate detected over the MCL are evaluated, or a HQ of 1 if the maximum concentration of nitrate is evaluated for potential exposures through ingestion of groundwater. A HQ of unity indicates that there is a potential for adverse health effects. Because of the conservative assumptions used in the evaluation, however, the estimate of a HQ of 1 may be an overestimation of the actual hazard. Trichloroethane is present in the groundwater at concentrations that are estimated to have a ICR of 3E-05 (based on 95-percent UCL) or 4E-05 (based on maximum concentration) if lifetime exposures were to occur through ingestion of groundwater and inhalation of volatiles from groundwater use in the home.

Of the COPC specified, three are classified as volatile contaminants that would generally be evaluated via the inhalation of volatiles from soil. These are tetrachloroethane, trichloroethane, and 1,1,1-trichloroethane. Although the inhalation of volatile contaminants from soil was suggested as a potential exposure pathway. EPA directed [Einan, 1992 (see appendix I)] indicated that data generated from soil gas surveys should not be used in risk assessment. Because the majority of the volatile COPC from the specified subunits have only been detected in soil gas the potential exposures and associated risks are not quantitatively evaluated in the BRSRA. The volatilization of contaminants from soil is qualitatively addressed in chapter 5, paragraph 5.4.

Lead exposures, as directed by EPA [Einan, 1991 and Einan, 1992 (see appendix A)] are evaluated using the UBK model. The UBK model predicts blood-lead levels in children from potential exposure to lead through soil ingestion, dust inhalation, and dietary exposures. Lead exposures are evaluated at the 1100-3 subunit and HRL.

Recreational pathways associated with the Columbia River are also evaluated in the BRSRA. The two pathways considered are the dermal contact with potentially contaminated water through swimming and the ingestion of fish caught from the Columbia River.

Other pathways may also occur that have not been evaluated in the BRSRA, however, the potentially dominant risk driving pathways and those routinely evaluated for residential scenarios are included.

Given the above considerations, the BRSRA identifies and evaluates the contaminants that are most likely to pose a potential human health risk if residential use of the subunits

were to occur. The COPC that pose a potential for noncarcinogenic systemic toxic effects (*i.e.*, HQ > 1) or ICR of >1E-06 for each subunit are discussed below.

6.4 1100-1 SUBUNIT

6.4.1 BISRA

Arsenic and vanadium are the COPC at this subunit. The HI (0.008) and total ICR (4E-07) do not exceed unity or 1E-06, respectively, for the subunit. Consequently, potential worker exposures to the maximum detected COPC would not be likely to result in adverse health effects.

6.4.2 BRSRA

(Does not Apply)

6.5 1100-2 SUBUNIT

6.5.1 BISRA

Chromium is the only contaminant of potential concern at this subunit. The HI (0.001) and the total ICR (1E-07), for the subunit do not exceed unity or 1E-06, respectively. Consequently, potential worker exposures to the maximum detected concentration of chromium would not be likely to result in adverse health effects.

6.5.2 BRSRA

Tetrachloroethane is the only contaminant of potential concern at this subunit. Residential exposure to the concentrations of tetrachloroethane detected at this subunit are not likely to result in adverse health effects. The subunit HI for the exposure pathways evaluated is 0.00003 and the ICR is 7E-09 (table 5-1). Based on the BRSRA, no contaminants of concern are identified at 1100-2.

6.6 1100-3 SUBUNIT

6.6.1 BISRA

The contaminant of potential concern at the 1100-3 subunit is chromium. The HI (0.0008) and total ICR (8E-08) for this subunit do not exceed unity or 1E-06, respectively.

Therefore, adverse systemic health effects are not likely for industrial workers exposed to the maximum concentration of contaminants detected at this subunit.

6.6.2 BRSRA

Arsenic, chromium, and lead are the COPC at this subunit. The ICR for exposure to arsenic at this subunit is 9E-06, primarily due to the potential ingestion of arsenic-contaminated soil. This estimate, however, includes the contribution of potential risk from the background concentration of arsenic in the soil. The ICR for the inhalation of fugitive dust containing chromium is less than 1E-06. All individual HQ's and the HI for the subunit are less than unity.

An evaluation of lead using the UBK model indicates that children exposed to lead in the soil and ingestion of garden produce potentially contaminated with lead will not result in blood-lead levels that exceed the currently recommended level of concern.

Based on this BRSRA, arsenic is the only possible contaminant of concern for the 1100-3 subunit. The ingestion of soil is the exposure pathway associated with the greatest estimated risk. However, the background concentration of arsenic normally present in soil is included in the risk estimate and may contribute significantly to the overall ICR.

6.7 1100-4 SUBUNIT

6.7.1 BISRA

Arsenic and beryllium are the only two COPC identified at the 1100-4 subunit. The HI (0.006) for this subunit is less than unity. Therefore, adverse systemic health effects are not likely for industrial workers exposed to the maximum concentration of contaminants detected at this subunit.

Although individual contaminant ICR's are all negligible (*i.e.*, $<$1E-06), the soil ingestion pathway and subsequently the subunit-specific ICR is 1E-06. However, because the site of the subunit is located inside a building and under a cement floor, this risk estimate is considered to be an overestimation of actual risk. Hence, potential worker exposure to the maximum detected concentrations of contaminants are not likely to result in any adverse health effects.

6.7.2 BRSRA

(Does not Apply)

6.8 UN-1100-6 SUBUNIT

6.8.1 BISRA

BEHP, chlordane, and heptachlor are the COPC at this subunit. The HI (0.4) for this subunit is less than unity. Therefore, adverse systemic health effects are not likely for industrial workers exposed to the maximum concentration of contaminants detected at this subunit.

BEHP is the only COPC associated with ICR's greater than 1E-06. The ICR's for BEHP for the soil ingestion pathway, the fugitive dust pathway, and the dermal exposure pathway are 3E-05, 3E-08, and 3E-06, respectively, with a total ICR of 3E-05. Therefore, BEHP is the only contaminant of concern identified at the UN-1100-6 subunit.

An estimation of the ICR's for BEHP and chloride using the 95-percent UCL indicates no significant difference when compared to the ICR's estimated for the maximum detected concentrations. The ICR's for BEHP based on the 95-percent UCL for the soil ingestion pathway, the fugitive dust pathway, and the dermal exposure pathway are 2E-05, 2E-08, and 2E-06, respectively. The ICR's for chlordane based on the 95-percent UCL for the soil ingestion pathway, the fugitive dust pathway, and the dermal exposure pathway are 2E-07, 2E-10, and 2E-07, respectively. The total ICR for UN-1100-6 subunit is 3E-05 based on the maximum detected concentration, and 2E-05, based on the 95-percent UCL (see table 6-1).

6.8.2 BRSRA

The evaluation of potential exposures to BEHP and chlordane present in the soil at this subunit are associated with risks greater than 1E-06. The subunit HI for all pathways (table 6-1) is 23 (based on maximum contaminant concentrations) or 18 (based on the 95-percent UCL) indicating the potential for adverse systemic health effects in individuals that may ingest the soil from the site or eat produce grown at the site.

The subunit ICR is 3E-03 based on the maximum contaminant concentration and 2E-03 based on the 95-percent UCL. As with the HI, the potential ingestion of garden produce contributes the majority of the risk. Because of the conservative assumptions used in deriving the SF's and the conservative assumptions utilized in estimating the uptake of BEHP by plants, the actual risk may be less than the estimated risk.

The contaminants of concern at this subunit are:

Noncarcinogenic Effects:

- BEHP - Soil ingestion, Garden Produce Pathway
- Chlordane - Garden Produce Pathway

Carcinogenic Effects:

- BEHP - Soil ingestion, Dermal Exposure, Garden Produce Pathway
- Chlordane - Soil Ingestion, Dermal Exposure, Garden Produce Pathway

6.9 EPHEMERAL POOL

6.9.1 BISRA

Chlordane, heptachlor, and PCB's are COPC at the Ephemeral Pool. The estimated HI (= 0.03) for this subunit is less than unity. Therefore, adverse systemic health effects are not likely for industrial workers exposed to the maximum concentration of contaminants detected at this subunit.

PCB's are the only contaminant of potential concern associated with ICR's greater than 1E-06. The ICR's for PCB's are 3E-05, 8E-08, and 3E-05 for the soil ingestion pathway, the fugitive dust inhalation pathway, and the dermal exposure pathway, respectively, with a total subunit ICR of 3E-05 based on all COPC evaluated using the maximum contaminant concentrations.

An estimation of the ICR's for PCB's using the 95-percent UCL indicates similar results when compared to the ICR's estimated for the maximum detected concentrations. The ICR's for PCB's based on the 95-percent UCL for the soil ingestion pathway, the fugitive dust pathway, and the dermal exposure pathway are 9E-06, 3E-08, and 1E-05, respectively, with a total subunit ICR of 2E-05 (see table 6-1). Therefore, PCB's are the only contaminant of concern for the Ephemeral Pool.

6.9.2 BRSRA

Chlordane and PCB's are the COPC at this subunit. The subunit total HI is 3.6 (maximum contaminant concentration) or 2.5 (95-percent UCL concentration), related primarily to potential exposures to chlordane through the garden produce pathway. PCB's are not quantitatively evaluated for systemic toxic effects through the ingestion pathway because there are no published toxicity values for noncarcinogenic effects.

The subunit ICR is 3E-03 based on the maximum contaminant concentration and 1E-03 based on the 95-percent UCL. In both cases the risk is primarily due to the potential ingestion of PCB's through the garden produce pathway. Both chlordane and PCB's are contaminants of concern for this subunit as summarized below:

Noncarcinogenic Effects:

- Chlordane - Garden Produce Pathway

Carcinogenic Effects:

- Chlordane - Soil Ingestion, Dermal Exposure, Garden Produce Pathway
- PCB's - Soil ingestion, Dermal Exposure, Garden Produce Pathway

6.10 HRL

Fourteen COPC have been identified at HRL. The subunit HI for all pathways is (0.2) less than unity. Therefore, adverse systemic toxic effects are not likely based on the assumptions and maximum detected concentrations evaluated for the subunit.

The following are COPC with individual pathway ICR's that exceeded 1E-06 based on maximum detected contaminant concentrations, and therefore may be associated with adverse carcinogenic effects:

- Chromium - Fugitive Dust Inhalation
- PCB's - Soil Ingestion, Dermal Exposure

The inhalation of fugitive dust is associated with the greatest ICR, with a pathway ICR of 3E-05 associated primarily with chromium at the maximum detected concentration. The assumption that the entire landfill is uniformly contaminated with the maximum concentration of chromium detected and that all chromium is chromium(VI) results in an overestimation of actual risk. When chromium is evaluated using a conservatively biased 95-percent UCL based on the area where the highest concentrations of chromium were detected, the ICR is estimated as 2E-06 (see table 5-10). This risk estimate, however, would overestimate actual risks for most of the landfill.

PCB's are associated with the greatest risks for the soil ingestion pathway and the dermal exposure pathway, with pathway ICR's of 6E-05 and 8E-05, respectively, at the maximum detected concentrations. When evaluating the potential risks for PCB's based on a 95-percent UCL determined from the areas of greatest PCB detection, the estimated ICR's for the soil ingestion and dermal contact pathways are reduced by approximately a third, although they still exceed 1E-06.

Therefore, chromium and PCB's are the only contaminants of concern identified at HRL.

6.10.1 BRSRA

The subunit HI for all pathways evaluated at HRL is 5.6 based on the maximum detected contaminant concentration and 1.2 based on the 95-percent UCL. The total ICR for the subunit is 7E-03 based on the maximum detected concentration and 3E-03 for 95-percent UCL. The COPC identified at this subunit are:

Noncarcinogenic Effects:

- Nitrate - Groundwater Ingestion (however, nitrate would not be considered a contaminant of concern based on the 95-percent UCL contaminant concentration).

Carcinogenic Effects:

- Arsenic - Soil Ingestion
- Beryllium - Soil Ingestion, garden produce
- PCB's - Soil Ingestion, Dermal Exposure, Garden Produce Pathway
- Trichloroethane - Groundwater ingestion
- Chromium - Fugitive Dust Inhalation

6.11 RESIDENTIAL-RELATED RECREATIONAL PATHWAYS

As indicated in table 5-6, it is unlikely that adverse effects from exposure to trichloroethane would occur in residents who may swim in the Columbia River or ingest fish caught in the Columbia River. As discussed in chapter 3, trichloroethane may migrate to the river via the groundwater. The ICR's for both potential exposures are estimated as less than 4E-10.

6.12 SUMMARY

6.12.1 BISRA

The contaminants of concern for the individual subunits in the 1100-EM-1 Operable Unit as determined in this BISRA are:

- UN-1100-6 subunit
  BEHP

- Ephemeral Pool
  PCB's

- HRL
  Chromium
  PCB's

As discussed above, this list does not consider background conditions, and has been developed based on conservative industrial exposure parameters. The list of contaminants of concern assume that current land and water use for the 1100 Area will remain the same in the future and that onsite industrial workers are the primary receptors.

6.12.2 BRSRA

The contaminants of concern for the individual subunits in the 1100-EM-1 Operable Unit, based on this BRSRA, are:

- 1100-3

  Arsenic

- UN-1100-6 subunit

  BEHP
  Chlordane

- Ephemeral Pool

  Chlordane
  PCB's

- HRL

  Arsenic
  Beryllium
  Chromium
  Nitrate
  PCB's
  Trichloroethane

The BRSRA has been conducted at the direction of EPA. It should be noted that the residential use of any of the subunits at the 1100-EM-1 Operable Unit in the foreseeable future is improbable. The 1100-EM-1 Operable Unit is located in an area designated for industrial or commercial use, as zoned by the city of Richland. The subunits themselves are located immediately adjacent to actively used industrial sites such as the 1171 Building, railroad tracks, or parking lots. For HRL, it is also extremely unlikely that homes would be built immediately adjacent to the subunit, based on current regulations in the state of Washington. Consequently, the onsite residential exposure pathways that have been evaluated are not probable. As a result, the estimated risks presented in the BRSRA are very conservative estimates based on an unlikely scenario.

7.0 REFERENCES

Amdur, M.O., J.D. Doull, and C.D. Klaassen, editors, 1991, *Casarett and Doull's Toxicology: The Basic Science of Poison, 4th Edition*, Pergamon Press, New York, New York.

Baskett, R.L., 1983, *Fugitive Dust Emission Factors for the Mining Industry*, American Mining Congress, Washington, D.C.

Brady, N.C., 1974, *The Nature and Property of Soils*, 8th Edition, MacMillan Publishing Co., Inc., New York.

Clayton, G.D. and F.E. Clayton (eds.), 1981, *Patty's Industrial Hygiene and Toxicology*, John Wiley and Sons, New York, New York.

Cowherd Jr., C., G.E. Muleski, P.J. Englehart, and D.A. Gillette, 1985, *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites*, EPA/600/8-85/002, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C.

DOE/RL-90-18, 1990, Phase I Remedial Investigation Report for the Hanford Site; 1100 EM-I Operable Unit, DOE/RL 90-18, U.S. Department of Energy, Hanford Site, Richland Operations Office, Richland, Washington.

DOE/RL-90-32, 1991, *Phase I and II Feasibility Study Report for the Hanford Site 1100 EM-I Operable Unit*, DOE/RL 90-32, Rev. 1, U.S. Department of Energy, Hanford Site, Richland Operations Office, Richland, Washington.

DOE/RL-91-45, 1992, *Hanford Site Baseline Risk Assessment Methodology*, DOE/RL 91-45, Rev. 1, U.S. Department of Energy, Richland, Washington.

Einan, D., Environmental Protection Agency (EPA), (letter to R. Stewart, DOE-RL), May 30, 1991.

Einan, D., EPA, (letter to R. Stewart, DOE-RL), January 16, 1992.

EPA, 1988, *Superfund Exposure Assessment Manual*, EPA-540/1-88/001, U.S. Environmental Protection Agency, Washington, D.C.

EPA, 1989a, *Risk Assessment Guidance for Superfund: Volume 1, Human Health Evaluation Manual, Part A; Interim Final*, EPA/540/1-89/002, U.S. Environmental Protection Agency, Washington, D.C.

EPA, 1989b, *Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites*, OSWER 9355.4-02, U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington D.C.

EPA, 1991, *Health Effects Assessment Summary Tables: Annual FY-1991*, OERR 9200.6-303(91-1), U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C.

EPA, 1992a, *Integrated Risk Information System*, Access date: March, 1992, United States Department of Health and Human Services, National Library of Medicine Toxicology Data Network (TOXNET), Bethesda, Maryland.

EPA, 1992b, *Health Effects Assessment Summary Tables: Annual FY-1992*, OHEA ECAOCIN-821, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C.

EPA, 1992c, *Dermal Exposure Assessment: Principles and Applications*, EPA/600/8-91/O11B, U.S. Environmental Protection Agency, Exposure Assessment Group, Washington, D.C.

EPA-10, August 16, 1991, *Supplemental Risk Assessment Guidance for Superfund*, U.S. Environmental Protection Agency, Region 10, Seattle, Washington.

Kabata-Pendias, A. and H. Pendias, 1984, *Trace Elements in Soils and Plants*, CRC Press, Inc., Boca Raton, Florida.

Life Systems, Inc., 1989, *Toxicological Profile for Di(2-ethylhexyl)phthalate*, ATSDR/RP88/15, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

Ryan, E.A., E.T. Hawkins, B. Magee, and S.L. Santos, 1987, "Assessing Risk from Dermal Exposures at Hazardous Waste Sites," *Superfund 1987: Proceedings of the 8th National Conference*, Hazardous Materials Control Institute, Silver Springs, Maryland.

Shu, H.P., P. Teitelbaum, A.S. Webb, L. Marple, B. Brunck, D. DeiRossi, F.J. Murray, and D. Paustenbach, 1988, "Bioavailability of Soil-Bound TCDD: Dermal Bioavailability in the Rat," *Fundamental and Applied Toxicology*, 10:335-343.

Skidmore, E.L., and N.P. Woodruff, 1968, *Wind Erosion Forces in the United States and Their Use in Predicting Soil Loss*, Agriculture Handbook No. 346, U.S. Department of Agriculture, Agricultural Research Service.

U.S. Weather Bureau and SCS, 1962, *Evapotranspiration Maps for the State of Washington*, U.S. Weather Bureau, Office of the State Climatologist, Seattle, Washington.

Wisness, S., DOE-RL, letter to P. Day, EPA and T. Nord, State of Washington, Department of Ecology, October 30, 1991.

Woodruff, N.P., and F.H. Siddoway, 1965, *Wind Erosion Equations*, Soil Science Society of America, Proceedings, Vol. 29, pp 602-608.

APPENDIXES

This page left intentionally blank.

APPENDIX I

LETTERS AND MEMORANDA

This page left intentionally blank.

United States
Environmental Protection
Agency

Region 10
Hanford Project Office
712 Swift Boulevard, Suite 5
Richland WA 99352

EPA

May 30, 1991

Robert K. Stewart
Unit Manager
U.S. Department of Energy
P.O. Box 550, A6-95
Richland, Washington 99352

Re: 1100-EM-1 Remedial Investigation

Dear Mr. Stewart:

This letter has several purposes. First, the enclosure to this letter should provide the clarifications requested by the U.S. Department of Energy (DOE) on February 28, 1991 (Ref: Letter, S. Wisness to P. Day).

Secondly, due to other commitments (e.g. Tri-Party Agreement negotiations) and internal communication problems, combined EPA and Ecology comments on the "Remedial Investigation Phase 2 Supplemental Work Plan for the Hanford Site 1100-EM-1 Operable Unit" and the "Phase I and II Feasibility Study Report for the Hanford Site 1100-EM-1 Operable Unit" will be delayed by not more than 30 days. I expect to send any comments on the above documents by June 28, 1991.

Finally, in response to the April 26, 1991 letter from Mr. Wisness to Mr. Day, I understand and agree that interim milestones M-15-01B (November 1991) and M-15-01C (April 1992) are in jeopardy. I will work with you to develop an aggressive and attainable schedule upon which to develop a change package.

If you have any questions, please call me at 376-3883.

Sincerely,

David R. Einan
Unit Manager

cc: R. Hibbard, Ecology
J. Stewart, USACE
T. Veneziano, WHC
Administrative Record (1100-EM-1)

This page left intentionally blank.

CLARIFICATION OF 1100-EM-1 REMEDIAL INVESTIGATION ISSUES RAISED BY U.S. DEPARTMENT OF ENERGY

May 24, 1991

1. Identification of land areas for agricultural/residential land use exposure scenarios

The 1100-EM-1 subunits that should be included in the evaluation of risk from residential exposures for the baseline risk assessment are shown in Table 1. The rationale for including or excluding each subunit is also presented in Table 1.

The quantitative risk assessment of a residential scenario will provide risk estimates that are protective of agricultural health threats because an exposure pathway including homegrown vegetables and fruits is required. Agricultural scenarios do not need to be included in the baseline risk assessment. In addition, the health risk to agricultural workers is adequately addressed in the industrial scenario as provided in the baseline risk assessment (U.S. DOE, 1990).

2. Residential exposure scenario for the 1100-EM-1 baseline risk assessment

A residence should be located directly adjacent to each subunit. For the Horn Rapids Landfill, the residence should be placed near monitoring well MW-12.

Receptor populations should include typical populations such as children, adults, and the elderly.

Table 2 shows each subunit with its associated contaminants, exposure medium, and exposure routes. The information presented in Table 2 assumes that exposure to contaminants includes the following pathways: inhalation of vapors and particulates, accidental ingestion of soil, ingestion of homegrown vegetables and fruits, ingestion of drinking water, dermal contact with potable water, inhalation of vapors during showering, and dermal contact with soil.

The existing data are sufficient for performing residential risk assessments for the subunits listed in Table 1. All subunits should address exposure pathways related to contaminated soil. The Horn Rapids Landfill, however, should also address exposure pathways related to groundwater. Potential groundwater health threats will be assessed for the other subunits in the Phase 2 remedial investigation if the data support the need to do so.

It is appropriate to present the residential risk assessment in the baseline risk assessment uncertainty section.

3. Reasonable maximum exposure

A table summarizing exposure parameters used, references for those parameters, and rationales for using each parameter should be included as part of the baseline risk assessment.

Example calculations for one contaminant in each pathway should be provided in an appendix. The appendix should include generic equations as well as example calculations.

Reasonable maximum exposure parameters as outlined in Region 10 guidance (U.S. EPA, 1990a) should be used. If Region 10 guidance for a particular parameter is not published or established, then reasonable maximum exposure parameters as provided by U.S. Environmental Protection Agency (EPA) headquarters (1989) should be used. If U.S. EPA guidance is not available, then exposure parameters found in open literature or developed using professional judgment should be used.

4. Toxicity screening

The preliminary toxicity screening first compares contaminant concentrations to background, then to calculated toxicity screening criteria. The first step is acceptable. The second step may eliminate chemicals that individually may not pose a health risk, but cumulatively might pose health risks. Not enough information is provided by U.S. Department of Energy (U.S. DOE) (1990) to determine this. A table summarizing critical effects for all potential contaminants of concern before implementing the screening should have been provided in the Phase 1 remedial investigation report. Therefore, the acceptability of the screening method cannot be determined at this time.

Based on available information, the preliminary toxicity screening contains the following technical flaws:

- It appears that the screening criteria for lead is an applicable or relevant and appropriate requirement (ARAR). It is not appropriate for screening purposes to use an ARAR. Therefore, lead should be included in the baseline risk assessment.

- The use of the Occupational Safety and Health Administration's permissible exposure limit for polynuclear aromatic hydrocarbons (PAHs) as the surrogate residential exposure limit development is not appropriate. U.S. EPA (1990b) recommends that critical toxicity values for benzo(a)pyrene be used in the absence of critical toxicity values for PAHs. However, because the sampled PAH level exceeds the surrogate residential exposure limit screening criterion, and PAHs were not eliminated from the risk assessment at that point, it is not necessary to develop a new preliminary toxicity screening criterion for PAHs based on benzo(a)pyrene information. In the future, the critical toxicity values for benzo(a)pyrene should be used for developing a PAH toxicity screening criterion.

TABLE 1
RATIONALE FOR INCLUDING 1100-EM-1 SUBUNITS
RESIDENTIAL SCENARIO RISK ASSESSMENT

Subunit	Decision to Include	Rationale
Battery Acid Pit (1100-1)	No	The lead found in soil samples is most likely associated with backfill materials. The arsenic levels are not significantly elevated above background levels.
Paint and Solvent Pit (1100-2)	Yes	Significant levels of tetrachloroethene was found in soil. (Tetrachloroethene may also pose a groundwater health threat, but inclusion in the risk assessment will be based on Phase 2 RI results.)
Antifreeze and Degreaser Pit (1100-3)	Yes	Significant levels of lead, arsenic, and chromium were found in surface soil. (Chromium may pose a groundwater health threat, but inclusion in the risk assessment will be based on Phase 2 RI results.)
Antifreeze Tank Site (1100-4)	No	The tank has been removed and the subunit is located in a building. (Alpha and beta radiation may pose groundwater health threats, but inclusion in the risk assessment will be based on Phase 2 RI results.)
Radiation Contamination Incident (UN-1100-5)	No	Contamination no longer exists.

TABLE 1
RATIONALE FOR INCLUDING 1100-EM-1 SUBUNITS
RESIDENTIAL SCENARIO RISK ASSESSMENT
(Continued)

Subunit	Decision to Include	Rationale
Discolored Soil Site (UN-1100-6)	Yes	Significant levels of BEHP and chlordane were found in surface soil. (1,1,1-trichloroethane may pose a health threat in groundwater, but inclusion in the risk assessment will be based upon Phase 2 RI results.)
Horn Rapids Landfill	Yes	Significant levels of arsenic, chromium, lead, PCBs, tetrachloroethene, trichloroethene, and 1,1,1-trichloroethane were found in soil. Trichloroethane was found in groundwater. (Tetrachloroethene and 1,1,1-trichloroethane may pose groundwater health threats, but inclusion in the risk assessment will be based on Phase 2 RI results.)
Pit 1	No	Pit 1 is an operational gravel pit.
Ephemeral Pool	Yes	Significant levels of PCB and chlordane were found in surface soil.

Definitions:

RI = Remedial Investigation
BEHP = Bis(2-ethylhexyl)phthalate
PCB = Polychlorinated biphenyl

TABLE 2
EXPOSURE MEDIA AND EXPOSURE ROUTES FOR 1100-EM-1
RISK ASSESSMENT RESIDENTIAL SCENARIO

Subunit	Contaminants of Concern	Exposure Medium	Exposure Route IHL	ING	D
Paint and Solvent Pit (1100-2)	Tetrachloroethene	Soil	C		
Antifreeze and Degreaser Pit (1100-3)	Arsenic	Soil	C,S	C,S	C,S
	Chromium	Soil	C	S	S
	Lead	Soil	S	S	
Discolored Soil Site (UN-1100-6)	BEHP	Soil	C	C,S	C,S
	Chlordane	Soil		C,S	C,S
Horn Rapids Landfill	Arsenic	Soil	C,S	C,S	C,S
	Chromium	Soil	C	S	S
	PCB	Soil	C	C	C
	Tetrachloroethene	Soil	C		
	Trichloroethene	Soil	C		
		Groundwater	C	C	
	1,1,1-Trichloroethane	Soil	S		
	Lead	Soil	S	S	
Ephemeral Pool	Chlordane	Soil		C,S	C,S
	PCB	Soil		C	C

Definitions:

IHL = Inhalation
ING = Ingestion
D = Dermal
C = Exhibits carcinogenic effects in exposure route indicated
S = Exhibits systemic noncarcinogenic effects in exposure route indicated
BEHP = Bis(2-ethylhexyl)phthalate
PCB = Polychlorinated biphenyl

This page left intentionally blank.

REFERENCES

U.S. DOE, August 1990. Phase 1 Remedial Investigation Report for the Hanford Site 1100-EM-1 Operable Unit. U.S. Department of Energy, Richland, WA.

U.S. EPA, December 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A), Interim Final. EPA/540/1-89/002. U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA, January 1990. Statement of Work RI/FS Risk Assessment Deliverables. U.S. Environmental Protection Agency, Region 10, Seattle, WA.

U.S. EPA, September 1990. Slope Factors for PAHs (American Cross Armory, Chehalis, WA). Memo from Pei-Fung Hurst, Chemical Mixtures Assessment Branch, to Dana Davoli, Environmental Services Division, U.S. Environmental Protection Agency, Region 10, Seattle, WA.

This page left intentionally blank.

United States
Environmental Protection
Agency

Region 10
Hanford Project Office
712 Swift Boulevard, Suite 5
Richland WA 99352

EPA

January 16, 1992

Robert K. Stewart
Unit Manager
U.S. Department of Energy
P.O. Box 550, A5-19
Richland, Washington 99352

Re: 1100-EM-1 Risk Assessment

Enclosed please find the additional clarifications requested by the U.S. Department of Energy in regards to the above subject. These clarifications were also informally transmitted to you by cc:Mail. Also transmitted informally was a copy of a Groundwater Risk Assessment for Hanford 1100-EM-1 Operable Unit, Richland, Washington prepared for the U.S. Environmental Protection Agency (EPA) by PRC Environmental Management, Inc. This document is provided to you for information, especially as an example for formatting the revised baseline risk assessment for 1100-EM-1.

If you have any questions, please call me at (509) 376-3883.

Sincerely

David R. Einan
Unit Manager

cc: D. Lacombe, PRC
R. Hibbard, Ecology (w/ Risk Assessment)
W. Greenwald, USACE
Administrative Record, 1100-EM-1

This page left intentionally blank.

ATTACHMENT 2

1100-EM-1 ON-SITE RESIDENTIAL BASELINE RISK ASSESSMENT ISSUES

What is the role of the Hanford Site Baseline Risk Assessment Methodology (DOE-RL-91-45)? If the residential scenario from DOE-RL-91-45 is used, we should have EPA specify which pathways will be evaluated at each operable subunit.

EPA RESPONSE

The 1100-EM-1 residential risk assessment should use the residential scenario and associated pathways presented in the Hanford Site Baseline Risk Assessment Methodology (DOE 1991).

The residential scenario should be used for the 1100-EM-1 operable unit. The residential scenario was originally chosen because 1) it is the most conservative, 2) residences are in close proximity, and 3) industrial zoning is not a permanent remedial solution. In addition, an agricultural worker scenario was not requested because the remedial investigation report (DOE 1990) dealt sufficiently with that type of risk.

Although the agricultural scenario as defined in DOE (1991) is the most conservative, an agricultural scenario does not need to be considered at this time because farm dwellings are not the typical residences in the immediate area.

1. GROUNDWATER QUESTIONS:

According to the May 30, 1991, EPA letter, the only groundwater contaminant to be evaluated under an on-site residential scenario is trichloroethene (TCE) at the Horn Rapids Landfill with a residence and water supply well located at MW-12.

a. Will we be considering other potential groundwater contaminants at the Horn Rapids Landfill? At least two additional rounds of groundwater monitoring data are now available. When this information is evaluated it may identify other contaminants of potential concern or may confirm that TCE is not a contaminant of potential concern attributable to the landfill.

b. Do we consider groundwater contaminants related to Siemens/ANF activities? This would be especially important for radionuclides, nitrates, and TCE. If specific radioactive isotopes are not yet available from the sampling, it may be prudent to defer evaluation of such substances until the Phase II

RI rather than make too many conservative assumptions at this time.

c. Do we assume groundwater use despite the fact that city service exists to industrial, commercial, and residential areas in the vicinity of 1100-EM-1?

d. There is a conflict between State law and the suggested location of the residence with respect to the Horn Rapids Landfill. Do we assume the presence of a drinking water well even though WAC 173-160-205(2) does not permit location of such a well within 1000 feet of solid waste landfills? A possibility may be that the site of the potential residence is moved at least 1000 feet from the landfill thus limiting the potential contact with Horn Rapids Landfill contamination by other pathways.

EPA RESPONSE TO ITEM 1

a. The additional rounds of groundwater data should be evaluated. If the data indicate that contaminants other than trichloroethene are of concern (e.g. nitrate), those contaminants should be included in the risk assessment.

b. The risk assessment should consider contaminants related to Siemens/ANF activities because the issue is to understand the potential human health and environmental risks posed by the 1100-EM-1 operable unit irrespective of the original contaminant source. The risk assessment should focus only on chemical contaminants until adequate data is available for radionuclides.

c. The risk assessment should assume groundwater use.

d. The risk assessment should assume a drinking water well is located adjacent to the Horn Rapids Landfill. For an intrusion scenario, 1000 feet will not make much of a difference.

2. EXPOSURE PATHWAYS:

a. Are the residential exposure pathways only those outlined in Section 2, p. 1 of the May 30 letter? Should potential contamination of City of Richland water from groundwater reaching the Columbia River be considered? Where are the activities occurring for the pathways? (e.g., see 4a. and 5a. below)

b. Given the size of the landfill, the restricted area, and the distribution of the potential contaminants, what specific

assumptions should be made regarding access to the landfill by the assumed family located in the residence near the landfill (e.g., if the residence is located at MW-12)?

EPA RESPONSE TO ITEM 2

a. The risk assessment should include the exposure pathways as outlined in EPA (1991a), Section 2, page 1. In addition, the risk assessment should include additional contaminants or exposure pathways if new data indicate the need to do so.

   The risk assessment should consider the impact of groundwater on the Columbia River and the city of Richland well field.

b. Unrestricted access to the landfill should be assumed in the risk assessment because closure cannot be assumed at this time.

3. TOXICITY VALUES:

a. Should we assume that all toxicity values be updated to current values?

b. What RFD and slope factor should be used for lead?

EPA RESPONSE TO ITEM 3

a. Current toxicity values from the Integrated Risk Information System (IRIS) or the Health Effects Summary Tables (HEAST) should be used in the risk assessment.

b. Since no reference dose or slope factor is available for lead, the risk assessment should use the EPA Uptake/Biokinetic Model for determining site-specific risks from exposure to lead (EPA 1991b,c). The model predicts blood lead levels in the most sensitive population (children) via inhalation or ingestion.

4. EXPOSURE PARAMETERS:

What exposure parameters should be used? The May 30, 1991, letter recommends outdated reasonable maximum exposure parameters and does not consider new national standard default exposure parameters recommended in OSWER Directive 9285.6-03, March 25,

1991. In addition, Region-10 now recommends new parameters in the EPA Region 10 Supplemental Risk Assessment Guidance for Superfund, August 16, 1991.

EPA RESPONSE TO ITEM 4

Current exposure parameters as specified by EPA headquarters or Region 10 should be used in the risk assessment.

5. HOME GROWN FRUITS AND VEGETABLES:

a. Where should gardens be located? Are supposed on-site residences to be placed directly adjacent to the subunits? Are the gardens on the subunits? Since some subunits are small, could all of the subunit be garden thus limiting any regular child exposure to the dirt?

b. What specific fruits and vegetables should be evaluated?

c. What bioaccumulation factors should be used?

EPA RESPONSE TO ITEM 5

a. Dwellings should be located adjacent to the subunit.

The Exposure Factors Handbook (EPA 1990) gives the median size of a vegetable garden as 325 square feet (approximately 18 feet by 18 feet). Therefore, assume gardens are also located adjacent to the subunit.

Even if the entire subunit is garden, a child's exposure would not be limited because a garden is not an impermeable cover.

b,c. The following strategy is presented for the selection of fruits and vegetables:

- Three plant categories should be included in the risk assessment: root, fruit, and leafy vegetable.
- The bioaccumulation factor for the contaminants of concern should be determined for the three categories listed above.
- At least one plant from each category should be included in the risk assessment. Additional

plants may be included based on information obtained from Pao, et al. (1982) or other informational sources.

The references below may be useful in locating bioaccumulation factors:

- A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides Through Agriculture. C.F. Base, R.D. Sharp, A.L. Sjoreen, and R.W. Shore. ORNL-5786. Oak Ridge National Laboratory. 1984.

- Bioconcentration of Organics in Beef, Milk, and Vegetation. 1988. C.C. Travis and A.D. Arms. Environmental Science and Technology 22: 271-274.

6. CONTAMINANT CONCENTRATIONS:

a. Will the residential scenario consider Model Toxics Control Act definition of surface soils as a depth of 15 ft?

b. Additional soil gas data are available for Horn Rapids Landfill, UN-1100-6, and the South Pit. Should these data be incorporated in the on-site residential risk assessment?

c. Additional soil data are available for Horn Rapids Landfill and the Ephemeral Pool. Should these data be incorporated?

EPA RESPONSE TO ITEM 6

a. The residential scenario should consider the Model Toxics Control Act (Ecology 1991) definition of surface soils as a depth of 15 feet inasmuch as the risk assessment needs to consider accessible soil contaminant concentrations. If it is determined that the site needs cleanup to residential levels, then the surface soil depth of 15 feet should be used in the calculation of cleanup levels.

b. Soil gas surveys are used for field screening. Data generated from soil gas surveys should not be used in the risk assessment.

c. Any available soil data should be evaluated. If the data indicate contaminants are of concern, those contaminants should be included in the risk assessment.

7. TIME OF FUTURE SCENARIO:

When should the residential scenario be applied, (i.e., now, 30 years in the future, etc.)?

EPA RESPONSE TO ITEM 7

- For the purposes of the "residence" located adjacent to the Horn Rapids Landfill, the time period should be now, i.e. use the concentrations found in the well.

REFERENCES

DOE 1990. Phase 1 Remedial Investigation Report for the Hanford Site 1100-EM-1 Operable Unit. DOE/RL-90-18. August 1990. U.S. Department of Energy.

DOE 1991. Hanford Site Baseline Risk Assessment Methodology. Decisional Draft. DOE/RL-91-45. September 1991. U.S. Department of Energy.

Ecology 1991. The Model Toxics Control Act Cleanup Regulations. Chapter 173-340 WAC. Department of Ecology. February 28, 1991.

EPA 1990. Exposure Factors Handbook. EPA 600/8-89/043. U.S. Environmental Protection Agency. March 1990.

EPA 1991a. 1100-EM-1 Remedial Investigation. Letter from Dave Einan, U.S. Environmental Protection Agency. May 30, 1991.

EPA 1991b. Update on OSWER Soil Lead Cleanup Guidance. Office of Solid Waste and Emergency Response. August 29, 1991. U.S. Environmental Protection Agency.

EPA 1991c. User's Guide for Lead: A PC Software Application of the Uptake/Biokinetic Model Version 0.50, First Draft. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, U.S. Environmental Protection Agency. January 1991.

Pao, E.M., C.H. Fleming, P.M. Guenther, and S.J. Mickle. 1982. Foods Commonly Eaten by Individuals: Amount Per Day and Per Eating Occasion. Consumer Nutrition Center, Human Nutrition Information Service, U.S. Department of Agriculture, Hyatteville, MD 20782. Home Economics Research Report No. 44. March 1982.

DOE/RL-92-67

Department of Energy
Richland Operations Office
P.O. Box 550
Richland, Washington 99352

OCT 30 1991

91-ERB-202

Mr. Paul T. Day
Hanford Project Manager
U. S. Environmental Protection Agency
712 Swift Boulevard, Suite 5
Richland, Washington 99352

Mr. Timothy L. Nord
Hanford Project Manager
State of Washington
Department of Ecology
Mail Stop PV-11
Olympia Washington 99504-8711

Dear Messrs. Day and Nord:

LAND USE/RISK ASSESSMENT FOR THE 1100-EM-1 OPERABLE UNIT (OU)

The purpose of this letter is to inform you that the DOE Field Office, Richland (RL), will comply with direction as provided by your letters regarding a revised baseline risk assessment for the 1100-EM-1 OU as discussed with you earlier. Specifically, we will perform the required risk assessment that evaluates residential and agricultural scenarios as directed by the letter of January 23, 1991, from Mr. Dave Einan, U.S. Environmental Protection Agency (EPA) to Mr. Bob Stewart, RL and the follow-up clarifications contained in the May 30, 1991, letter from Mr. Einan to Mr. Stewart.

Whether to conduct a baseline risk assessment for the 1100-EM-1 OU using residential and agricultural scenarios has been a long term issue. RL continues to believe that neither residential nor agricultural use is reasonably likely in the areas within the OU. Further we do not believe that the risk assessment is necessary nor appropriate under applicable requirements of the National Contingency Plan or regulatory guidance.

Particularly troublesome aspects of the proposed risk assessment are the assumed exposure pathways for groundwater. Neither RL nor Siemens Nuclear Power Corporation (Siemens) believes that there is any reasonable expectation for either residential or agricultural use of the groundwater which may have been influenced by contamination from the Horn Rapids Landfill and/or Siemens. We continue to believe that the risk assessment contained in the completed Remedial Investigation (RI) Phase 1 Report used appropriate Reasonable Maximum Exposure (RME) assumptions.

Notwithstanding our position, to show good faith in providing the requested information and to get on with the scheduled RI/ Feasibility Study (FS), RL will perform a revised baseline risk assessment as discussed in the first paragraph of this letter. However, our agreement to proceed with this assessment is premised on the understanding that we have reached agreement with EPA and the State of Washington Department of Ecology (Ecology) on two points:

1. That RL's performance of a revised baseline risk assessment, which includes evaluation of residential and agricultural scenarios, shall not be viewed as a concession by RL or be used as any evidence that residential or agricultural use of the property or groundwater is reasonable or foreseeable; and

2. That RL has the right under the Hanford Federal Facility Agreement and Consent Order (Tri-Party Agreement) to take to dispute and to obtain dispute resolution of any future regulatory direction to evaluate or investigate remedial alternatives based on assumed residential or agricultural use of the property or groundwater.

Because the EPA has the lead responsibility for the 1100-EM-1 OU, we have discussed these two points with you. We understand that both EPA and Ecology are in agreement with RL. This letter confirms those discussions. If we have misunderstood in any way the agencies' views, please inform us in writing within ten days of the date of this letter.

RL has begun to work on the revised baseline risk assessment. It is expected that the work to perform this assessment can be accomplished in about two months. However, we have not evaluated factors associated with the slightly elevated alpha or beta contamination in the groundwater and this could modify the amount of time required. Work on the assessment for these scenarios will be completed without prejudice to RL's right to express reservations about the accuracy of the assessment and the sufficiency of available data to support a meaningful assessment.

We have discussed with you a proposed procedure to bring any dispute over land use in the 1100-EM-1 OU to early formal Dispute Resolution. We have agreed that RL will provide the revised baseline risk assessment to EPA/Ecology promptly upon its completion. In transmitting the assessment, RL will request regulatory direction regarding the land use scenario(s) on which to base remedial alternatives selection for inclusion in the FS. After you have received the assessment, we request a response to this request as quickly as practicable.

Messrs. Day and Nord -3- OCT 3 0 1991
91-ERB-202

It is our understanding that neither EPA nor Ecology has determined that remedial alternatives must be based in whole or in part on the assumption of residential or agricultural use of the land or groundwater. Thus, a dispute over land use assumptions may not develop. However, if EPA directs RL to develop remedial alternatives based on assumed residential or agricultural use or uses of the property or groundwater, then we will immediately invoke the dispute resolution process under the Tri-Party Agreement for a resolution of the appropriate land use assumptions.

Regarding the land use issue as discussed in the May 30, 1991, letter from Mr. Einan to Mr. Stewart, two technical issues related to the baseline risk assessment were carried forward; i.e., calculation of RME and use of the Golder toxicity screening technique. It is our understanding that these issues have been satisfactorily resolved through informal discussions and exchanges of information among the Unit Managers and respective support contractors. If these issues have not been resolved to your satisfaction, please communicate such to Mr. Stewart.

Should you have any questions about this letter, please call Mr. Bob Stewart on (509) 376-6192.

Sincerely,

Steven H. Wisness
Hanford Project Manager

ERD:RKS

cc:
D. Einan, EPA
L. Goldstein, Ecology
W. Greenwald, USACE
M. Harmon, EM-442
R. Hibbard, Ecology
G. Hofer, EPA
M. Lauterbach, WHC
R. Lerch, WHC
C. Malody, Siemens
T. Nord, Ecology
J. Stewart, USACE
G. Welch, Siemens,
(Law Dept. Bellevue, WA)
T. Wintczak, WHC
S. Woodbury, EH-222
T. Veneziano/L. Powers, WHC
Administrative Record, 1100-EM-1,
H4-22

This page left intentionally blank.

Script for Superfund Technical Support Center Questions
on
Tetrachloroethylene, Trichloroethylene and Styrene

Tetrachloroethylene (perchloroethylene, PERC)

The carcinogenicity characterization has a long history. A July 1985 Health Assessment Document for Tetrachloroethylene (Perchloroethylene), EPA # 600/8-82/005F, classified the agent in Weight-of-Evidence Group "C - Possible Human Carcinogen" mentioning that this would be reevaluated because of new information. The 1985 document also provided upper bound inhalation and oral risk estimates. An April 1987 Addendum to the Health Assessment Document, EPA# 600/8-82/005FA, proposed that the Weight-of-Evidence be upgraded to "B2 - Probable Human Carcinogen" and provided a revised inhalation risk estimate. A February 1991 document titled Response to Issues and Data Submissions on the Carcinogenicity of Tetrachloroethylene, EPA# 600/6-91/002A discussed newer data relative to weight-of-evidence classification. The Agency's Science Advisory Board has reviewed these documents finding them to be technically adequate while offering an opinion that the weight-of-evidence is on C-B2 continuum (C=Possible Human Carcinogen, B2=Probable Human Carcinogen). At present time, the Agency has not adopted a final position on the weight-of-evidence classification.

The upper bound risk estimates from the 1985 Health Assessment Document as amended by updated inhalation values from the 1987 Addendum have not as yet been verified by the IRIS-CRAVE Workgroup. The estimates are viewed as useful information in the context of the information available in the 1985-1987 period.

ORAL: 1985 HAD; Unit risk = 1.5E-6 per ug/L

Slope Factor = 5.2E-2 per mg/kg/day

INHALATION: 1987 Addendum; Unit risk = range form 2.9E-7 to 9.5E-7 with a geometric mean of 5.8E-7 per ug/cu.m

Slope factor = 2.0E-3 per mg/kg/day

Those needing to make a choice about carcinogenicity have found the 1985, 1987 and 1991 EPA documents and the 1988 and 1991 Science Advisory Board letters of advice useful background information. When the Agency makes a decision about weight-of-evidence, the CRAVE-IRIS verification will be completed and the information put on IRIS.

Trichloroethylene (TCE)

The current phase of the carcinogenicity characterization for trichloroethylene started with a July 1985 Health Assessment Document for Trichloroethylene, EPA# 600/8-82/006F which classified trichloroethylene in Weight-of-Evidence Group "B2 - Probable Human Carcinogen". Inhalation and oral upper bound risk estimates were provided. This information was verified on IRIS from 3/87 through 7/89. A June 1987 Addendum to the Health Assessment Document for Trichloroethylene, EPA# 600/8-82/006FA proposed that the Weight-of-Evidence finding of "B2" was further supported by newly available animal bioassay data and offered a minor revision to the inhalation upper bound risk estimate. In 1988 the Agency's Science Advisory Board offered an opinion that the weight-of-evidence was on C-B2 continuum (C=Possible Human Carcinogen, B2=Probable Human Carcinogen). The Agency withdrew the IRIS carcinogenicity file in 7/89 and has not adopted a current position on the weight-of-evidence classification.

The quantitative risk estimates provided in the 1985 Health Assessment Document and 1987 Addendum have been reviewed by the IRIS-Crave Workgroup but are not verified as such pending resolution of the weight-of-evidence classification. The upper bound risk values in these documents are as follows:

ORAL: 1985 HAD; Unit Risk = 3.2E-7 per ug/L
Slope Factor = 1.1E-2 per mg/kg/day

INHALATION: 1987 Addendum; Unit Risk = 1.7E-6 per ug/cu.m.
Slope Factor = 6.0E-3 per mg/kg/day

When the Agency adopts a current position on weight-of-evidence classification, the trichloroethylene file will be reentered on IRIS.

APPENDIX II

TOXICITY PROFILES

This page left intentionally blank.

1.0 TOXICITY PROFILES

The purpose of appendix II is to present toxicological information used in the BISRA and BRSRA. Tables II-1 and II-2 present toxicity values for all contaminants evaluated in chapter 2. This appendix provides toxicity profiles for potential contaminants of concern identified at the 1100-EM-1 Operable Unit and carried through the risk assessment. This information supplements information discussed in chapter 4.0. The categories of information include:

- General background information
- Exposure routes
- Acute toxicity
- Chronic toxicity
- Carcinogenicity
- Toxicity values and supporting information.

Data sources for the information provided in the appendix are from EPA documents and standard reference texts. These sources are:

- EPA Integrated Risk Information System (IRIS)
- EPA Health Effects Assessment Summary Tables (HEAST)
- SRC, Toxicological Profile for Individual Compounds, Agency for Toxic Substances and Disease Registry (ATSDR)
- Casarett and Doull's Toxicology, The Basic Science of Poisons (Amdur et al. 1991)
- Patty's Industrial Hygiene and Toxicology (Clayton and Clayton, 1981)
- Threshold Limit Values and Biological Exposure Indices
- 29 CFR 1910.1000
- Recommendations for Occupational Safety and Health Standards.

1.1 ANTIMONY

Elemental antimony does not exist naturally in the environment, but is found in small amounts as part of the earth's crust. Antimony has been detected in air, water and soil in varying concentrations. Soil concentrations are usually less than 1 μg/kg. Concentrations up to 2550 μg/kg have been found at antimony-processing sites. Antimony at these sites is strongly attached to the soil.

Exposure to antimony can occur through inhalation of antimony-containing particles, ingestion of antimony-containing soils, and ingestion of foodstuffs containing antimony. It is not known if contamination through dermal contact with antimony-containing soils is a route of exposure.

Table II-1. Summary of Noncarcinogenic Toxicity Information for Contaminants at the 1100-EM-1 Operable Unit. (sheet 1 of 3)

Contaminant	ORAL						INHALATION					
	Oral Rfd (mg/kg-day)	Oral Rfd (basis/source)	Confidence Level	Critical Effect	Uncertainty Factors	Modifying Factors	Inhalation Rfd (mg/kg-d)	Inhalation Rfd (basis/source)	Confidence level	Critical effect	Uncertainty Factor	Modifying Factor
Antimony	4E-4	Water/IRIS	low	longevity, blood gluc.	1,000	1	-	-	-	-	-	-
Arsenic	3E-4	Food/IRIS	medium	hyperpigmentation keratosis	3	1	-	-	-	-	-	-
Barium	7E-2	Water/IRIS	medium	incr. blood press	3	1	1E-4	HEAST	-	-	1,000	-
Beryllium	5E-3	Water/IRIS	-	none observed	100	1	-	-	-	-	-	-
Cadmium	1.0E-03	Food/IRIS	high	significant proteinuria	10	1	-	-	-	-	-	-
Chromium VI	5E-3	Water/IRIS	low	none	500	1	-	-	-	-	-	-
Cobalt	6E-02	EPA Region 10	-	-	-	-	-	-	-	-	-	-
Copper	4E-02	EPA Region 10	-	GI irritation	-	-	-	-	-	-	-	-
Lead	ND	-	-	-	-	-	ND	-	-	-	-	-
Manganese	1E-1	Food/IRIS	medium	CNS effects	1	1	1.1E-04	IRIS	-	CNS and resp. symptom	300	3
Mercury (in organic)	3E-4	HEAST	-	kidney effects	1,000	-	8.5E-05	HEAST	-	-	30	-
Nickel	2E-2	Food/IRIS	medium	decrease body + organ weight	300	1	-	-	-	-	-	-
Selenium	5.3E-3	IRIS	-	hair + nail loss	3	1	-	-	-	-	-	-
Silver	5E-3	I.V./IRIS	low	argyria	3	1	-	-	-	-	-	-
Thallium	7E-5	-/IRIS	-	SGOT and serum LDH level	3,000	-	-	-	-	-	-	-

Table II-1. Summary of Noncarcinogenic Toxicity Information for Contaminants at the 1100-EM-1 Operable Unit. (sheet 2 of 3)

Contaminant	ORAL						INHALATION					
	Oral Rfd (mg/kg-day)	Oral Rfd (basis/source)	Confidence Level	Critical Effect	Uncertainty Factors	Modifying Factors	Inhalation Rfd (mg/kg-d)	Inhalation Rfd (basis/source)	Confidence level	Critical effect	Uncertainty Factor	Modifying Factor
Vanadium	7E-3	Water/HEAST	.	none	100	.	.	.	.	.	.	.
Zinc	2E-1	HEAST	.	anemia	10	.	.	.	.	.	.	.
BEHP	2E-2	IRIS	low	liver weight	1000	1	.	.	.	.	.	.
Beta-HCH (Hexachlorocyclo-hexane)	.	.	.	.	.	.	.	.	.	.	.	.
Chlordane	6E-5	Food/IRIS	low	liver hypertrophy in mice	1,000	1	.	.	.	.	.	.
Chlorobenezene	2E-2	Food/IRIS	medium	liver changes	1,000	1	5E-3	HEAST	.	liver, kidney effects	10,000	.
Cyanide	2E-2	Food/IRIS	medium	weight loss, thyroid effect, myelin deg.	100	5	.	.	.	.	.	.
DDT	5E-4	Food/IRIS	medium	liver lesions	100	1	.	.	.	.	.	.
Endosulfan II	5E-5	Diet/IRIS	medium	kidney toxicity	3,000	1	.	.	.	.	.	.
Endrin	3E-4	Diet/IRIS	low	mild changes liver	100	1	.	.	.	.	.	.
Heptachlor	5E-4	Food/IRIS	low	liver weight	300	1	.	.	.	.	.	.
2-Hexanone	.	.	.	.	.	.	.	.	.	.	.	.
Naphthalene	4.0E-02	Gavage/HEAST	medium	decreased weight	1,000	.	.	.	.	.	.	.
PCBs	.	.	.	.	.	.	.	.	.	.	.	.
Tetrachloroethene	1E-2	Gavage/IRIS HEAST 1991	medium	hepatotoxic in mice, weight gain rat	1,000	1	.	.	.	.	.	.

Table II-1. Summary of Noncarcinogenic Toxicity Information for Contaminants at the 1100-EM-1 Operable Unit. (sheet 3 of 3)

Contaminant	ORAL						INHALATION					
	Oral Rfd (mg/kg-day)	Oral Rfd (basis/source)	Confidence Level	Critical Effect	Uncertainty Factors	Modifying Factors	Inhalation Rfd (mg/kg-d)	Inhalation Rfd (basis/source)	Confidence level	Critical effect	Uncertainty Factor	Modifying Factor
1,1,1-Trichloroethane	9E-2	Oral/HEAST	-	-	1,000	-	3E-01	Oral/HEAST	-	-	1,000	-
Trichloroethane	-	-	-	-	-	-	-	-	-	-	-	-

Sources: Integrated Risk Information System Access: March 1992a
Health Effects Assessment Summary Tables (1992b) unless otherwise indicated
ND Not Determined
- Not Available

Table II-2. Summary of Carcinogenic Toxicity Information for Contaminants at the 1100-EM-1 Operable Unit. (sheet 2 of 3)

Contaminant	Weight of Evidence Classification	Type of Cancer	Oral SF $(mg/kg\text{-}d)^{-1}$	Oral SF (source)	Inhalation SF $(mg/kg\text{-}d)^{-1}$	Inhalation SF (source)
Beta-HCH (Hexachlorocyclo-hexane)	C	-	1.8E+0	IRIS	1.8E+0	IRIS
Chlordane	B2	-	1.3E+0	IRIS	1.3E+0	IRIS
Chlorobenezene	-	-	-	-	-	-
Cyanide	-	-	-	-	-	-
DDT	B2	-	3.4E-1	IRIS	3.4E-1	IRIS
Endosulfan II	-	-	-	-	-	-
Endrin	-	-	-	-	-	-
Heptachlor	B2	-	4.5E+0	IRIS	4.5E+0	IRIS
2-Hexanone	-	-	-	-	-	-
Naphthalene	-	-	-	-	-	-
PCBs	B2	-	7.7E+0[a]	IRIS	7.7E+0[b]	Surrogate
Tetrachloroethene	B2[c,d]	-	5.2E-2	Region-10[c]	2E-03	Region-10[c]
1,1,1-Trichloroethane	-	-	-	-	-	-

Table II-2. Summary of Carcinogenic Toxicity Information for Contaminants at the 1100-EM-1 Operable Unit. (sheet 1 of 3)

Contaminant	Weight of Evidence Classification	Type of Cancer	Oral SF $(mg/kg\text{-}d)^{-1}$	Oral SF (source)	Inhalation SF $(mg/kg\text{-}d)^{-1}$	Inhalation SF (source)
Antimony	-	-	-	-	-	-
Arsenic	A	Skin, Lung	1.75E+0[a]	Surrogate	5.0E+1	IRIS/HEAST
Barium	-	-	-	-	-	-
Beryllium	B2	-	4.3E+00	IRIS	8.4	HEAST
Cadmium	B1	-	ND	-	6.1E+0	IRIS/HEAST
Chromium VI	A	Lung	ND	-	4.1E+01	IRIS/HEAST
Cobalt	-	-	-	-	-	-
Copper	-	-	-	-	-	-
Lead	B2	-	ND	-	ND	-
Manganese	-	-	-	-	-	-
Mercury (in organic)	-	-	-	-	-	-
Nickel	A	Lung	-	-	8.4E-1	IRIS
Selenium	-	-	-	-	-	-
Silver	-	-	-	-	-	-
Thallium	-	-	-	-	-	-
Vanadium	-	-	-	-	-	-
Zinc	-	-	-	-	-	-
BEHP	B2	-	1.4E-02	IRIS	1.4E-02[c]	Surrogate

Table II-2. Summary of Carcinogenic Toxicity Information for Contaminants at the 1100-EM-1 Operable Unit. (sheet 3 of 3)

Contaminant	Weight of Evidence Classification	Type of Cancer	Oral SF $(mg/kg\text{-}d)^{-1}$	Oral SF (source)	Inhalation SF $(mg/kg\text{-}d)^{-1}$	Inhalation SF (source)
Trichloroethene	B2[c,d]		1.1E-02	Region-10[c]	6.0E-03	Region-10[c]

[a]Based on proposed arsenic unit risk of 5E-05 $\mu g/L$ (EPA 1991)
[b]Surrogate; assumed same as oral SF
[c]As recommended by Superfund Technical Support Center, April 1992 (EPA-10, Personal Communication)
[d]Weight of evidence classification under review
ND Not determined
- Not available
Sources: Integrated Risk Information System Access: March 1992a
Health Effects Assessment Summary Tables (1992b unless otherwise indicated)

Antimony has been used in medical treatments for persons infected with parasites. Exposure to antimony for prolonged periods can cause eye, skin, and respiratory irritations. Other reported antimony-related symptoms include heart problems, vomiting, and diarrhea. The carcinogenicity and teratogenicity of antimony are currently unknown. High concentrations have caused animal mortality but it is not known if this would occur in humans. Human health effects (heart problems and stomach ulcers) have been observed following exposure to airborne antimony at a concentration of 2 mg/m^3 for 8 to 24 months. Lung, eye, and skin irritations were present following 9 years of exposure to 9 mg/m^3 of antimony.

Data from acute exposure indicate that the gastrointestinal (GI) tract is a target organ following inhalation of antimony. Respiratory and cardiovascular effects also occur, but at exposure levels lower than those associated with gastrointestinal effects. The GI tract is also targeted following oral exposure to antimony. There is no information on target organs following dermal exposures.

Chronic exposure to antimony indicates that the respiratory tract, heart, eyes, and skin are target organs. There is no evidence of increased cancers due to chronic airborne antimony exposure by humans. Studies have shown that chronic oral exposures result in accumulation of antimony in the liver and GI tract. No dermal cancer studies were located in the literature.

The EPA has set an oral chronic reference dose (RfD) of 4E-04 mg/kg-d (IRIS) for antimony with an uncertainty factor of 1000. The RfD confidence is low due to a lack of adequate oral exposure investigations. The critical effects in humans include altered blood chemistry, reduced longevity and changes in the blood glucose level.

The Occupational Health and Safety Administration (OSHA) has set a limit of 0.5 mg/m^3 of antimony in workroom air during an 8 hour time-weighted average (TWA). The National Institute of Occupational Health and Safety (NIOSH) recommends an identical limit. The American Conference of Governmental Industrial Hygienists (ACGIH) has a Threshold Limit Value (TLV) of 0.5 mg/m^3.

1.2 ARSENIC

Arsenic is a common element found in the earth's crust usually in the form of arsenic bearing minerals. It is difficult to characterize as a single element because its chemistry is very complex. Elemental (metallic) arsenic is a relatively non-toxic steel gray metal which is fairly rare in nature. Trivalent and pentavalent forms are widely distributed in nature as both inorganic and organic compounds. The trivalent form is more toxic than the pentavalent form, and the inorganic is typically more toxic than the organic form which is rapidly eliminated. Soil levels range from 1 to 50 mg/kg, but are usually less than 10 mg/kg. In the soil, compounds revert to arsenates which are held by clay soils and are not readily available for plant uptake. Arsenic compounds have found use as pesticides, herbicides, wood preservatives, pigments, and medicinal agents.

Depending on the chemical species, arsenic can be toxic via all routes of exposure. Acute arsenic poisoning is usually the result of homicidal, suicidal, or accidental ingestion of inorganic arsenical. Arsenic is well absorbed from the gastrointestinal tract. Symptoms include constriction of the throat, stomach pain, vomiting, fever, cardiac disturbances, and watery diarrhea usually within 4 h of exposure. If the amount is sufficiently high (100 to 200 mg), death as a result of severe fluid loss and shock may occur within 24 to 48 h. Toxicity in humans and animals results from the interaction of arsenic with sulfhydryl groups in essential proteins.

Chronic exposures can produce toxic reactions in the skin, mucous membranes, gastrointestinal tract (GI) tract, and central nervous system (CNS). Peripheral vascular disease (gangrene) related to a cumulative effect can occur. Liver injury has also been associated with chronic exposure. Arsenic has a predilection for skin and concentrates in hair and nails. Long term exposure to arsenic compounds can result in hyperpigmentation, hyperkeratosis (thickening, drying, and cracking of the skin and growth of warts), and skin cancer. Skin cancer has been primarily associated with ingestion of drinking water containing high levels of arsenic. Chronic exposure through inhalation of arsenic compounds can produce weakness, loss of appetite, nausea, occasional vomiting and diarrhea, and lung cancer.

The oral RfD for arsenic provided in HEAST is 3E-04 mg/kg-d and the adverse effects of concern are keratosis and hyperpigmentation. Arsenic is a confirmed human carcinogen (EPA weight-of-evidence-classification Group A) known to produce lung cancer from inhalation and skin cancer from ingestion of drinking water. The inhalation slope factor (SF) listed in IRIS is 5.0E+01 $(mg/kg\text{-}d)^{-1}$ and based on excellent exposure assessment, using air monitoring and some biomonitoring, in large populations of smelter workers. The carcinogenic risk associated with ingestion of inorganic arsenic has been the focus of much debate. A mean unit risk of 0.00005 $(ug/L)^{-1}$ has been recommended by EPA (IRIS, EPA 1992). The unit risk is defined as the risk associated with a lifetime consumption of inorganic arsenic in drinking water. Applying standard exposure assumptions, this unit risk corresponds to an estimated oral slope factor of 1.75 $(mg/kg\text{-}d)^{-1}$. This proposed value, reflecting the most recent opinions regarding the mechanism of action of ingested inorganic arsenic, is used to assess the carcinogenic oral exposure to arsenic. The unit risk is based on human studies showing a definite dose-response relationship between the consumption of drinking water and the development of skin cancer.

Recommended occupational air exposure limits of arsenic are also available. The ACGIH has established a TWA TLV of 0.2 mg/m^3 (as arsenic) for arsenic and soluble compounds, in situations other than the production of arsenic. OSHA has established a TWA permissible exposure limit (PEL) of 0.1 mg/m^3 for inorganic arsenic (29 CFR 1910.1018). The National Institute for Occupational Safety and Health (NIOSH) recommended exposure limit (REL) is 0.002 mg/m^3.

1.3 BARIUM

Barium is a silvery-white metal that occurs in nature in many different forms. It is found naturally in drinking water and food. Barium and barium compounds are commonly used in various industries and in human health care. For example, barium carbonate, barium chloride and barium hydroxide are used to make ceramics, pesticides and additives for oil and fuels. Barium sulfate is used by medical doctors for medical tests and X-ray photography. There is limited quantitative information regarding the extent of barium absorption following inhalation, oral or dermal exposure; however, as with other metals, barium is probably very poorly absorbed from gastrointestinal tract.

Occupational studies of workers exposed to barium dust have shown that workers have developed "baritosis". Affected workers did not show any clinical symptoms except a significantly higher incidence of hypertension (*i.e.*, high blood pressure). The most commonly observed cardiovascular effects in cases of acute ingestion of barium compounds are hypertension and abnormalities in heart rhythm, while respiratory weakness and paralysis is observed in cases of acute ingestion of barium salts by humans. Acute exposure in rats indicates a lethal dose$_{50}$ (LD_{50}) of 132 mg/kg-d for adult rats and 220 mg/kg-d for weanlings.

The EPA has set an RfD of 0.05 mg/kg-d for chronic oral exposures. Confidence in the oral RfD is medium. Increases in blood pressure have been observed as a critical effect in oral exposure studies. An inhalation RfD of 1E-04 mg/kg-d was derived by the EPA based on a short-term reproductive study in rats. This RfD is under review and subject to change as indicated in HEAST. There are no reliable data at present regarding the carcinogenicity of barium.

1.4 BERYLLIUM

Beryllium occurs in nature in rocks, soils and volcanic dust. It does not occur in its elemental form naturally. Beryllium compounds vary in water solubility. A major portion of beryllium will bind to soil and is not likely to migrate deeper into the ground and groundwater.

The primary exposure routes for beryllium are inhalation and ingestion. The dermal route is a minor one. Most ingested beryllium (>99 percent) is excreted. Inhaled beryllium that enters the lungs remain there for an extended period of time (months to years). Beryllium contact with open wounds can cause rashes or ulcers. Acute airborne exposure to beryllium can result in lung damage similar to pneumonia. Hypersensitivity to beryllium can also result from exposures. Chronic exposure at levels permitted under OSHA may result in lung damage to some workers.

The EPA has set an oral RfD of 5E-03 mg/kg-d (IRIS) with an accompanying uncertainty factor of 100. The confidence is low due to limited toxicity data by the oral route. There are no toxic effects reported for the reference dose. Beryllium is a B2 (probable) human carcinogen. The human epidemiology studies are considered inadequate. The oral slope factor (SF) for beryllium is 4.3E+00 $(mg/kg\text{-}d)^{-1}$ (IRIS) based on water

ingestion, and the inhalation SF is 8.4E+00 $(mg/kg\text{-}d)^{-1}$ (IRIS). Both slope factors were derived from experimental animal exposures to beryllium sulfate and other beryllium compounds. Lung and bone cancer are the most common cancers associated with beryllium exposure.

Airborne 8 hour TWA workplace exposures have been set as follows: OSHA, 0.002 mg/m^3; NIOSH, Ca (carcinogen)-lowest feasible concentration is 0.00005 mg/m^3; ACGIH, 0.002 mg/m^3.

1.5 BIS (2-ETHYLHEXYL)PHTHALATE

Bis (2-ethylhexyl)phthalate (BEHP) is a chemical used to make plastics more flexible. This compound is a constituent of numerous products including rainwear, flooring, shower curtains, and medical tubing. This substance and other phthalate-ester plasticizers have been found to be general contaminants in virtually all soil and water ecosystems. Insoluble phthalate esters complex with fulvic acid components of humic substances in soil. Fulvic acid functions as a solubilizer for the phthalates and thus serves to mediate the mobilization and transport of phthalates in soil and water. The widespread occurrence of phthalates such as BEHP has produced concern regarding their toxicity.

BEHP is well absorbed orally and there is evidence of some absorption through the dermal and inhalation routes of exposure. Acute toxicity is low by all routes of exposure. No effects have been observed from a single 5,000 mg oral dose in humans while 10,000 mgs produced only some gastrointestinal distress.

Animal studies indicate the liver and testes are target organs for adverse effects from chronic exposure to BEHP. This compound has also been reported to affect male and female reproductive capacity and oral ingestion has produced birth defects in laboratory animals. The chronic oral RfD is 2E-02 (mg/kg-d). A 1953 study is cited by IRIS in which the observed critical effect was an increase in relative liver weight. Confidence in the RfD is low to medium. Although sufficient numbers of animals were tested and multiple endpoints measured, only two dosages were utilized for less than lifetime exposures to determine RfD. Corroborating chronic animal bioassays, however, do support this RfD.

BEHP is considered to be a probable human carcinogen (EPA B2 classification) based on a 1982 National Toxicology Program (NTP) oral study in animals. A statistically significant, dose-dependent increase in liver tumors was observed in male and female mice and female rates receiving BEHP in food. The oral SF listed in IRIS is 1.4E-02 $(mg/kg\text{-}d)^{-1}$. A potential source of variation in the NTP study that could effect the slope factor in the use of an intake based on standard food consumption rates rather than administration of a known dose. Evidence for carcinogenicity of BEHP in exposed human populations is inadequate.

The ACGIH recommended TLV-TWA for BEHP is 5 mg/m^3 and a TLV short-term exposure limit (STEL) of 10 mg/m^3. The NIOSH REL is reduction of the exposure to the lowest feasible concentration because of the cancer-causing potential of BEHP. The OSHA PEL for BEHP is 5 mg/m^3.

1.6 CHLORDANE

Chlordane is man-made chemical used prior to 1983 as an agricultural pesticide and until 1988 for termite control. It is a multicomponent mixture with alpha- and gamma-chlordane as the primary components. Exposure to chlordane is possible via all routes including inhalation, ingestion, and dermal absorption. Chlordane is extremely persistent in the environment and can be toxic to wildlife.

In humans, acute toxicity from inhaling high concentrations of chlordane vapors is manifested as headache, irritation, confusion, and gastrointestinal complaints. Similar adverse effects have occurred after dermal contact and heavily contaminated soil for several hours. Suicidal or accidental ingestion of large quantities of chlordane have produced liver damage, seizures, and death. The acute lethal dose in man is not known, but has been estimated to be 25 to 50 mg/kg by ingestion.

Chronic toxic effects have not been identified in workers who produced or used chlordane. The critical effects in a 1983 chronic feeding study in rats was regional liver hypertrophy in female rats. Based on this study, the EPA oral RfD reported in IRIS is 6E-05 (mg/kg-d). Confidence in this RfD, however, is low. The database lacks adequate reproductive studies, testing in multiple mammalian species, and inadequate assessment of sensitive endpoints. Chlordane is known to biaccumulate in body fat with chronic exposure.

The EPA classifies chlordane as a B2 probable human carcinogen. Results of a 1977 National Cancer Institute (NCI) study indicate a significant dose-response increase in liver tumors in mice. These data are supported by additional animal studies. However, evidence from human studies to document the carcinogenicity of chlordane is insufficient. An oral SF for chlordane of 1.3E+00 $(mg/kg\text{-}d)^{-1}$ is reported in IRIS. The inhalation SF published in IRIS is 1.3E+00 $(mg/kg\text{-}d)^{-1}$.

The OSHA established TWA-PEL for chlordane is 0.5 mg/m^3 with a recommendation to protect skin in order to limit dermal absorption of chlordane. This occupational exposure limit is the same as that recommended by NIOSH and the ACGIH.

1.7 CHROMIUM

Elemental chromium does not exist naturally in the environment, but is found primarily as a part of chromite ore. In compounds, this element exists in one of three valence states, +2, +3, or +6. The trivalent form is an essential human micronutrient involved in carbohydrate metabolism. Adverse effects have not been associated with the trivalent form. The hexavalent form is important industrially (typically in the form of chromates) and has been associated with serious toxicities.

Hexavalent chromium is mobile in soil, but under aerobic and acidic soil conditions, it is reduced to trivalent chromium which readily precipitates with carbonates, hydroxides, and sulfides in the soil. Hexavalent chromium is toxic to plants; however, plants actually

tolerate relatively high levels of chromium in the soil and do not bioaccumulate significant amounts.

Human toxicity has been associated with hexavalent chromium by all routes of exposure. Hexavalent chromium is irritating and short-term high exposures can result in adverse effects at the site of contact, whether it is the skin, GI tract, or respiratory tract. Such contact can result in coughing, wheezing, irritation and perforation of the nasal mucosa, and pulmonary edema. Kidney and liver damage have also been associated with acute exposures. Hexavalent chromium is a potent sensitizer causing allergic reactions in the lungs, nasal passages, and skin. Long term exposure to airborne hexavalent chromium higher than natural background levels is known to produce lung and respiratory tract cancer in humans.

The EPA has determined the oral RfD for hexavalent chromium as 5E-03 mg/kg-d (IRIS) based on a drinking water study in rats. The confidence in this RfD is low and no critical effects were observed because of poor study design.

Hexavalent chromium is classified by EPA as a known human carcinogen (weight-of-evidence classification is Group A) by inhalation exposure. A number of studies, cited in IRIS, demonstrate dose-response relationships between hexavalent chromium exposure, and lung tumor production. The inhalation SF is 4.1 E+01 $(mg/kg\text{-}d)^{-1}$. No evidence exists to indicate that chromium is carcinogenic by the oral route.

Occupational air exposure limits for chromium are based on the toxicities associated with different forms. For carcinogenic forms of hexavalent chromium, the NIOSH TWA recommendation is 0.001 mg/m^3. The OSHA Permissible Exposure Level (PEL) for chromium metal is 1 mg/m^3, and the Immediately Dangerous to Life or Health (IDLH) level is 500 mg/m^3.

1.8 COPPER

Copper is a naturally occurring metal in rock, soil, water, sediment, and air. It also occurs in plants and animals. Copper compounds are not easily removed from the environment. Copper is an essential element for all known living organisms and is important for iron utilization in humans.

Exposure to copper can occur through the inhalation, ingestion, and dermal routes. Copper in concentrations over 1 mg/L has been found in household water supplies that utilize copper pipes. Dietary intakes of naturally occurring copper-containing foods amount to about 1 mg/day.

Acute copper exposure by ingestion can cause vomiting and diarrhea. The copper is excreted after several days. It is unknown how much exposure by inhalation and dermal routes occurs. Chronic exposure to high concentrations of copper can cause eye, nasal and oral irritations, headaches, dizziness and diarrhea. Liver and kidney damage can occur following high intakes of copper. Copper exposure is not known to cause cancer.

Workers exposed to airborne copper experienced respiratory irritation, hepatomegaly (enlargement of the liver), and ocular mucosal irritation. Metal fume fever has been associated with exposure to copper fumes. Gastrointestinal effects include anorexia, nausea, and occasional diarrhea.

An interim oral RfD of 4.0E-02 mg/kg-d has been recommended for copper by the EPA. Gastrointestinal irritation is the critical effect associated with copper exposure. Copper is not classified as a carcinogen.

The OSHA, NIOSH, and ACGIH occupational exposure limit is 1 mg/m^3 as copper dust.

1.9 DDT

DDT is a synthetic chemical produced for control of pests on crops and control of insects that act a vectors for diseases such as malaria and typhus. The abbreviation stands for 1,1,1-trichloro-2,2-bis-(p-chlorophenyl)ethane. It was one of the most widely used pesticides in the world. Technical DDT is primarily composed of three forms (p,p'-DDT, o,p'-DDT and o,o'-DDT), which are white, crystalline, tasteless and almost odorless solids. In addition, 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) and 1,1-dichloro-2,2-bis (p-chlorophenyl)ethane (DDD) are found as contaminants and degradation products in technical DDT. DDT and its metabolites are persistent in the environment, bioaccumulate through the food chain and have been detected in human adipose tissues. The presence of DDT in the environment is generally as a result of past use of the insecticide and subsequent movement from sites of application to land, water and air.

The central nervous system is a major target organ in humans and animals; the liver is also a major target organ in animals. Occupational exposure by inhalation, skin absorption and dermal contact with liquid forms of DDT have shown some CNS effects such as cold moist skin, hypersensitivity to contact, tremor, and convulsions. The acute oral exposure in mice indicates lethal $dose_{50}$ (LD_{50}) that range from 237 to 325 mg/kg and in rats the LD_{50} range from 113 to 800 mg/kg. Doses as high as 285 mg/kg have been ingested accidentally by humans with no fatal results.

Chronic exposure of experimental animals to DDT is associated with tremors and general hyperirritability. In one human study, DDT exposure for 12 to 18 months at 0.61 mg/kg have induced hepatic, hematological and cardiovascular responses. The EPA has set an oral RfD of 0.0005 mg/kg and slope factors of 0.34 $(mg/kg\text{-}day)^{-1}$ for both oral and inhalation exposures (HEAST). DDT is classified as probable human carcinogen by EPA weight-of-evidence classification (Group B2) based on positive cancer findings in animal studies.

The OSHA occupational exposure limit TWA is 1 mg/m^3 with indication for potential dermal absorption. NIOSH regards DDT as a potential carcinogen and recommended the exposure limit of 0.5 mg/m^3. ACGIH recommended TWA for DDT of 1 mg/m^3.

1.10 HEPTACHLOR

Heptachlor is a synthetic chemical produced as a component of the pesticide chlordane (approximately 10 percent by weight). Heptachlor is metabolized to heptachlor epoxide by humans, animals and bacteria in the environment. The EPA has banned the use of heptachlor as an insecticide for crops, for homes and buildings; however, it is still approved to kill fire ants in power transformers.

Human exposure to heptachlor or heptachlor epoxide commonly occurs by ingestion of contaminated water or food, and may occur by inhalation and skin absorption. The target organ affected by heptachlor and heptachlor epoxide are the central nervous system and the liver (seen as changes in the enzymes and cells).

In humans, signs of neurotoxicity (irritability, salivation, lethargy, dizziness, labored respiration, muscle tremors, and convulsions) are seen following exposure to technical grade chlordane which contains between 6 to 30 percent heptachlor. However, these effects can not be attributed solely to heptachlor. Heptachlor and heptachlor epoxide have long half-lives and because they are lipophilic, they bioaccumulate in the adipose (fat) tissues. Measurable levels of heptachlor and heptachlor epoxide have been detected in breast milk and serum without evidence of adverse health effects. Acute exposure in rodents and rabbits indicate lethal dose$_{50}$ (LD_{50}) that range from 40 to 162 mg/kg for heptachlor and 39 to 144 mg/kg for heptachlor epoxide.

The EPA has set an oral RfD of 0.0005 mg/kg and a slope factor of 4.5 (mg/kg - day)$^{-1}$ for both oral and inhalation exposures (IRIS). Heptachlor is classified as probable human carcinogen by EPA weight-of-evidence classification (Group B2) based on positive cancer findings in animal studies.

Both OSHA and the NIOSH recommended occupational exposure on TWA basis of 0.5 mg/m^3. Only NIOSH regarded heptachlor as potential human carcinogen. The dermal absorption is likely and should be prevented as necessary. The ACGIH TWA for heptachlor is also 0.5 mg/m^3.

1.11 Beta-HEXACHLOROCYCLOHEXANE

Beta-Hexachlorocyclohexane (HCH) is a synthetic chemical that exists in eight isomers. All HCH isomers are solids at room temperature. Gamma-HCH, commonly called lindane, has been used a an insecticide on fruit vegetable and other crops, and to treat head and body lice in humans. The general population can be exposed to lindane, alpha-, beta-, and delta-HCH in the air surrounding heptachlor manufacturing plants or agricultural fields where the pesticide is used, and through ingestion of contaminated food and water. Production of lindane in the U.S. was prohibited by 1976, and none of the isomers are currently produced in the U.S. It is currently imported from France, Germany, Japan and China. The primary health effects associated with exposure to HCH are hematological, hepatic, renal, immunological, neurological, reproductive and cancer.

Most available information in the literature is for the health effects of lindane or gamma-HCH. Typically, humans are not exposed to the individual isomers of HCH but to gamma-HCH or to technical-grade HCH which contains several HCH isomers. In rats hematological effects, specifically reduced numbers of red and white blood cells, were observed in rats fed beta-HCH in diet for 13 weeks.

The EPA has set a SF of $(1.8\ mg/kg\text{-}d)^{-1}$ for inhalation exposure (IRIS). It is classified as possible human carcinogen (Group C) since there is a limited evidence of carcinogenicity in animals, and no published human data is available.

Both OSHA and the NIOSH recommended exposure limits TWA of $0.025\ mg/m^3$ for gamma-hexachlorocyclohexane. The ACGIH determines exposure limit TWA of $0.5\ mg/m^3$. Skin precautions are also recommended to prevent dermal absorption.

1.12 LEAD

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. It is widely distributed in the environment, and can be transported long distances. Anthropogenic sources of lead come from gasoline additives, various metal products, ammunition, paint, and storage batteries. The biggest single source of lead in air is from automobile exhaust. Oral exposure occur from ingestion of contaminated food and beverages, in addition to incidental soil ingestion.

Acute exposure data for inorganic lead inhalation are not available. The reported lethal concentration$_{50}$ (LC_{50}) in rats for inhalation of tetramethyl and tetraethyl lead are 8,870 and $850\ mg/m^3$, respectively.

Children and pregnant women are the most sensitive populations to chronic effects from lead exposure. In children lead exposure is associated with frequent ingestion of dirt (pica) and inhalation of household dust from crawling and playing on floors. The effects of such exposure are reported as a decrease in IQ, neurobehavior impairment and hearing problems. Excessive exposures can result in serious neurological effects including changes in brain function (encephalopathy) which may progress to coma. The mortality rate for untreated lead encephalopathy in children was approximately 65 percent prior to the introduction of chelation therapy.

Transplacental transfer of lead from mother to fetus in humans has been demonstrated in several studies. This transfer can result in nervous system damage or changes. Lead also interferes with heme biosynthesis by altering the activity of three aminolevulinic acid (ALAD) enzymes. The result is a reduction of hemoglobin concentration in blood (anemia).

Although EPA has classified lead as a B2 carcinogen (probable human carcinogen based on adequate animal studies) there are no Agency-verified toxicological values that can be used to perform a risk assessment and to develop protective soil cleanup levels for lead. Studies relating soil lead to blood lead levels are difficult to compare. However, EPA has

recommended soil criteria for lead, as an interim guideline (EPA, 1989b) of 500 to 1,000 ppm total lead to be protective of sensitive populations.

1.13 NICKEL

Nickel is a naturally occurring metal found in the earth's crust. Nickel can also be found in wind-blown soil. Many nickel compounds are water-soluble, causing the water to have a green color. Nickel is released into the environment during metal working processes, and incineration and power production. Nickel will settle into the soil where it has an affinity for iron- or manganese-containing particles. Under acidic conditions, nickel may migrate into groundwater. Nickel does not appear to bioaccumulate in fish or plants. Food naturally contains nickel, and adult dietary intake of nickel is estimated to be in the range of 300 - 600 μg/day.

Exposure routes for nickel include inhalation, ingestion and dermal contact. Inhaled particles can enter the bloodstream, if small, or remain in the lungs if large. Ingested nickel will enter the body through the stomach and intestines. Small amounts of nickel can enter the bloodstream through dermal contact. The kidneys are the primary target organ. Nickel is excreted through feces and to a lesser extent through urine. Excretion is nearly completed in 4 to 5 days.

Exposure to nickel has been shown to cause lung and nasal sinus cancers. The heart, blood, and kidneys have also been shown to be effected by exposure to nickel. Dermal exposures can result in skin rashes and asthma. Allergic contact dermatitis from exposure to nickel is common in persons in the general population. Nickel's reproductive effects are unknown.

The EPA has established an oral RfD of 2E-02 mg/kg-d (IRIS) based on food consumption. Decreased body and organ weights have been reported as the critical effects of nickel exposure. An inhalation RfD has not been determined. There is inadequate evidence for carcinogenicity by the oral route to support the establishment of an oral SF.

Airborne occupational exposure limits are: OSHA, 0.1 mg/m^3 for soluble compounds and 1 mg/m^3 for insoluble compounds; NIOSH, 0.015 mg/m^3, based on a determination that nickel refinery dust is a carcinogen; ACGIH, 1 mg/m^3 for both soluble and insoluble compounds. The ACGIH is currently reviewing its limits.

1.14 NITRATE

As a class, nitrate compounds are a variety of chemicals used as explosives, medications, dyes, food additives, and as numerous other industrial products. Nitrate occurs naturally, and the majority of dietary intake is from vegetables. The dietary contribution from drinking water is usually quite small. The nitrate form of nitrogen is very water soluble and is highly mobile in water and soil contributing to concern over the presence of these compounds in the environment.

Exposure can result primarily from ingestion of contaminated water, but may also be associated with ingestion of soils and dermal contact with contaminated media. Toxicity is related to the specific nitrate compound. However, as a class, acute exposure to nitrates can produce headache, decrease blood pressure, abdominal pain, dilation of blood vessels, and methemoglobinemia, an impaired ability of the blood system to transport oxygen. Chronic exposure may result in weakness, general depression, headache, and mental impairment.

Human toxicity to nitrates in water is due to the conversion of nitrate to nitrite which results in the oxidation of hemoglobin to methemoglobin. Animals are a poor model for methemoglobin formation because many species lack nitrate-reducing bacteria. Infants, however, are particularly susceptible to nitrates due to their high gut content of nitrate-reducing bacteria, their lower enzymatic capacity to convert methemoglobin back to hemoglobin, and the presence of hemoglobin F, which is more susceptible to oxidation.

The chronic RfD for nitrate as nitrogen is 1.6E+00 mg/kg-d based on human infant studies of exposure to nitrate in drinking water. The observed adverse effect was methemoglobinemia. No uncertainty factors have been applied to this intake because of the RfD was determined from epidemiological studies in the most sensitive human population. Thus, confidence in the RfD is high. Nitrate has not been evaluated for carcinogenic potential.

1.15 POLYCHLORINATED BIPHENYLS

Polychlorinated biphenyls (PCB's) are very stable materials that contain 12 to 68 percent chlorine and are extremely persistent in the environment. Because of their low flammability and stability, PCB's have been used as insulating materials in electrical transformers and capacitors, as plasticizers in waxes, in paper manufacturing, and for a variety of other industrial purposes. The diversity of their use patterns, the large quantities used, and their stability has led to widespread occurrence of these compounds in soil and water. PCB's have been banned from use in the U.S. since 1978, but are still found in older electrical equipment and as contaminants in the environment. All PCB's are mixtures of chlorinated congeners, but the exact nature of these mixtures is unknown. Arochlor 1260 and Arochlor 1254 are commonly recognized PCB products. The last two digits in the number indicate the percentage of chlorine in the compound (*i.e.*, 60 and 54 percent, respectively).

Exposure to PCB's can occur from inhaling PCB-contaminated particulates, dermal absorption, or ingestion of contaminated food, soil, or water. Toxicity by all routes of exposure is similar. However, because of high public awareness of PCB's, in large part due to their extensive publicity, concern about exposure may far outweigh documented human toxicity.

Skin irritation can occur with acute and chronic exposure. A severe and disabling form of acne called chloracne is the primary dermal effect. Chronic toxicity studies in animals have suggested that PCB's can cause respiratory tract impairment, neurotoxicity, liver damage, birth defects, and cancer. PCB congeners vary in their potency for producing

biological effects, but little is known about which congeners may be responsible for the effects and to what extent the effects occur in humans.

PCB's as a group (not as specific congeners or total chlorine mixtures) are considered probable human carcinogens because of data in animals indicating increased liver cancer in exposed animals. However, the commercial preparations used may not be representative of actual mixtures of congeners found in the environment. There is inadequate but suggestive evidence that PCB's may also cause liver cancer in humans by all routes of exposure.

The EPA oral SF listed in IRIS is 7.7 $(mg/kg\text{-}d)^{-1}$. This slope factor is based on a study in rats showing a sequential progression of liver lesions to liver cancer during the natural life of the rat. The EPA carcinogenicity classification for PCB's is B2 (probable human carcinogen).

Occupational exposure limits for polychlorinated biphenyls are based on the chlorine content of the compound. Both OSHA and the ACGIH recommend a TWA of 0.5 mg/M^3 for 54 percent PCB's. The NIOSH recommendation for an occupational exposure limit is 0.001 mg/M^3. This level is the minimum reliably detectable concentration using the recommended sampling and analytical methods. Skin precautions are also recommended to prevent dermal absorption.

1.16 TETRACHLOROETHENE

Tetrachloroethene, also known as perchloroethylene and tetrachloroethylene, is a nonflammable liquid solvent used for dry cleaning fabrics and for metal degreasing operations. When tetrachloroethene evaporates, it produces an ether-like odor. However, it is relatively resistant to hydrolysis and biodegradation and thus persists in the environment. Tetrachloroethene is moderately to highly mobile in soil and susceptible to significant leaching.

The primary route of exposure to tetrachloroethene is through inhalation of vapors. Ingestion may occur from contaminated water. Dermal absorption is limited because tetrachloroethene does not penetrate intact skin to any great extent. The principal target organs are the CNS, liver, and kidney. Acute exposure to tetrachloroethene in confined, poorly ventilated areas, can produce dizziness, headache, confusion, nausea, and difficultly in walking. These effects are rapidly reversed when the individual is moved to clean air. The effect of long-term, low level exposure is not as well understood. Studies in animals suggest liver and kidney damage, birth defects, leukemia, and liver cancer may occur.

The oral RfD provided in IRIS for tetrachloroethene is 1E-02 (mg/kg-d). The critical adverse effects found in animal studies used to determine the RfD were liver damage in mice and weight gain in rats. No inhalation RfD is currently available. Confidence in this RfD is only medium; a good overall database of information is available, but insufficient reproductive studies have been conducted.

The carcinogenicity of tetrachloroethene is under review as is the classification of B2 (probable human carcinogen) or C (possible human carcinogen). Currently, epidemiological studies suggest an association between tetrachloroethene and an increased cancer risk but the human studies provide no good quantitative exposure information and involve exposure to other chemicals. Thus, the association is inconclusive. All SF's have been withdrawn form IRIS. The Superfund Technical Support Center recommends an oral SF of 5.2E-02 $(mg/kg-d)^{-1}$ and an inhalation SF of 2.0E-3 $(mg/kg-d)^{-1}$.

1.17 THALLIUM

Thallium is a bluish-white metal that is widely distributed in trace amounts in the earth crust. It is present in air, water and soil. It can be found in pure form or mixed alloys with other metals. Thallium can also be found combined with other substances such as bromine, chlorine, fluorine and iodine to form salts. Thallous is the most common form of thallium in the environment. Manufacturing industries of electronic devices, switches, and closures are significant users of this metal. Thallium compounds have limited use in the manufacture of special glasses and for medical procedures that evaluate heart disease. Thallium was used as a rat poison until 1972 when it was banned in the U.S. because of its potential to cause adverse health effects in exposed human populations.

Human exposure to thallium may occur by inhalation, ingestion or dermal absorption. The general population is exposed most frequently by ingestion of contaminated foods. Thallium compounds such as thallium oxide and thallium sulfate can be lethal at relatively low doses; however, typical human exposure levels are significantly below such doses. Thallium compounds affect the respiratory, cardiovascular, GI, and CNS systems. They are also toxic to the liver, kidneys, and the male reproductive system. Temporary hair loss has also been associated with ingestion of thallium in human.

The EPA has set an oral RfD of 0.00007 mg/kg-d for chronic oral exposure. No published inhalation RfD is available. There are no reliable data at present regarding the carcinogenicity of thallium.

The OSHA, NIOSH and ACGIH each recommend an occupational exposure limit TWA of 0.1 mg/M^3. There is a potential for dermal absorption and should be prevented when necessary.

1.18 1,1,1-TRICHLOROETHANE

1,1,1-trichloroethane (TCA), also known as methyl chloroform, is a halogenated hydrocarbon used primarily as a solvent because of a favorable combination of chemical, physical, flammability, and toxicologic properties. Although TCA is probably the least toxic chlorinated solvent, careless use, high volatility, and poor disposal practices have contributed to the potential human and environmental exposure.

Acute exposure to TCA can produce CNS depression. Inhalation of 1,600 mg/M^3 by humans produces no untoward response while inhalation of 5,400 to 10,800 mg/M^3 for 60 min can produce eye and nasal irritation and minor CNS impairment. The inhalation of 100,000 mg/M^3 for 60 min can produce anesthesia, cardiac sensitization to epinephrine, and possible death. Chronic, industrial exposure to TCA over 6 years did not demonstrate liver toxicity or cardiac toxicity in humans. Some studies have suggested that animals exposed to TCA may develop fatty livers and liver necrosis. However, no adverse effects were detected in a 6-month inhalation study in guinea pigs on which the IRIS oral RfD is based. An older supporting study noted only slight growth retardation in chronically exposed animals. The oral RfD is 9.0E-02 (mg/kg-d). Confidence in the RfD is medium to low because the number of animals at each dose level was limited, lengths of exposure were variable, and few toxic endpoints were examined. No inhalation RfD is published in IRIS, but HEAST lists an inhalation RfD for TCA of 3.0E-01 (mg/kg-d). The adverse effect noted for the inhalation RfD is hepatotoxicity.

Animal studies have not demonstrated carcinogenicity nor are there any human data reported to indicate that 1,1,1-trichloroethane is a human carcinogen.

1.19 TRICHLOROETHENE

Trichloroethene (also known as trichloroethylene) is a colorless liquid with an odor similar to ether or chloroform. This chemical is a man-made solvent used for degreasing metal parts, extracting caffeine from coffee, and in numerous consumer products such as typewriter correction fluid, paint removers, and spot removers.

Trichloroethene moves readily through soil and groundwater. Ingestion of contaminated water and inhalation of volatilized trichloroethene are the chief source of exposure. Absorption is not significant from skin contact with this solvent.

Acute oral toxicity in humans is low. Death has occurred from an ingested dose of 170 mg/kg. Acute effects from inhalation of trichloroethene are associated with the central nervous system (dizziness, headache, sleepiness) and occur at a threshold of 436 to 592 mg/M^3. Extremely high, acute exposures may produce cardiac rhythm disturbances. In animals, chronic exposure to trichloroethene by inhalation and ingestion has produced liver and kidney damage and may affect reproductivity toxicity.

Neither IRIS nor HEAST currently provide an RfD for trichloroethene and determination of an RfD is pending. Trichloroethene may induce lung cancer in animals when inhaled and may produce liver cancer in animals from oral administration. The EPA weight-of-evidence classification of B2 (probable human carcinogen) is under review. The oral and inhalation SF's for trichloroethene have also been withdrawn from IRIS pending further review of carcinogenicity studies. The Superfund Technical Support Center recommends an oral SF of 1.1E-2 (mg/kg-d)$^{-1}$ and an inhalation SF of 6.0E-3 (mg/kg-d)$^{-1}$.

1.20 VANADIUM

Vanadium is a metal found in compounds that are widely distributed at low concentrations in the earth's crust. Elemental vanadium does not occur in nature, but is associated with over 50 different mineral ores and in fossil fuels. Vanadium replaces other metals such as iron, titanium and aluminum in crystal structures. Natural releases of vanadium to soil result from the weathering of rock-bearing vanadium minerals, precipitation/deposition of vanadium from the atmosphere or water, and plant and animal wastes. Anthropogenic sources of vanadium are: fossil fuel combustion, mining, slag heaps, sewage sludge, and certain fertilizers.

The only significant effect of vanadium exposure in human is mild to moderate respiratory distress, and mucosal irritation from exposure to vanadium dust. Workers exposed to vanadium through inhalation may develop coughs, chest pain, sore throat or eye irritation that can last for several days, following the exposure. These effects are not specific to pure vanadium but are equally associated with other vanadium chemical forms, following inhalation exposure.

The EPA has set an oral RfD of 0.007 mg/kg-d for chronic exposure via drinking water ingestion. An assessment of carcinogenic potential in humans can not be made at present because of the inadequacy of human and animal data.

The OSHA, NIOSH and ACGIH recommend the same occupational exposure limit TWA of 0.05 mg/m^3 as respirable dust and fume. NIOSH recommends a ceiling exposure level (REL) for 15 minutes.

1.21 ZINC

Zinc is a common element in the earth's crust. It is detected in rock, soil, groundwater, surface water, and air. Zinc may be released by natural or anthropogenic activities. Major anthropogenic sources are metallurgic wastes from smelter and refining operations, mining drainage, electroplating, smelting, plastics, agricultural practices, and industrial and municipal waste effluents.

Zinc is an essential nutrient and is found in all foods. The average American daily intake is 12 to 15 mg, mostly from food. Zinc is important for the maintenance of healthy skin and hair, good healing, and resisting infections. Zinc does not accumulate with continued exposure, but the body regulates absorption and storage depending on body needs. It is often concentrated in the tissues of organisms even in the absence of abnormally high background concentrations.

Overexposure to zinc by oral ingestion can produce severe gastric and digestive problems. Inhalation of zinc dust or fumes from smelting or welding induced a syndrome called metal fume fever, characterized by difficulty in breathing and flu-like symptoms. The degree of adverse effects appears to be influenced by the associated compounds in zinc salt or oxides. The EPA has set an oral RfD of 0.2 mg/kg-d. The critical effect of zinc

exposure is anemia. No published inhalation RfD is available. Currently zinc is not classified as a human carcinogen.

THIS PAGE INTENTIONALLY
LEFT BLANK

APPENDIX III

RISK ASSESSMENT CALCULATIONS

This page left intentionally blank.

This appendix presents the equations used to generate the Intake and Risk Assessment Tables created for the residential scenario risk assessment, but are similar to those used for the industrial scenario. All example calculations are based on the maximum contaminant concentration from the Phase I RI data, although the same calculations can be used with the 95 percent UCL concentrations.

1.0 CALCULATION OF CONTAMINANT INTAKES FOR THE SOIL INGESTION, INHALATION, AND DERMAL PATHWAYS

Standard EPA equations for calculation of intakes, as provided in RAGS (EPA, 1989a) and EPA (1991a) are used as the basis for all intake calculations. The basic equation for calculating intakes, normalized with respect to body weight, via soil ingestion or inhalation is:

$$\text{Intake} = \frac{C \times IR \times EF \times ED \times CF}{BW \times AT} \quad (1)$$

where:

Intake	=	chronic daily intake of the contaminant (mg/kg-d)
C	=	concentration of contaminant in the medium (*e.g.*, mg/kg or mg/m^3)
IR	=	intake rate (*e.g.*, mg/d or m^3/d)
EF	=	exposure frequency (d/yr)
ED	=	exposure duration (yr)
BW	=	body weight (kg)
AT	=	averaging time (d/yr x yr)
CF	=	conversion factor (as appropriate)

All exposure parameters (*i.e.*, body weight, averaging time, contact rate, exposure frequency, and exposure duration) are those presented for the residential scenario, as presented in EPA Region-10 guidance (EPA-10, 1991). A summary of the residential exposure factors is provided in table III-1.

Table III-1. Summary of Residential Scenario Exposure Factors.

Exposure Factor	Reasonable Maximum Exposure[a]
Intake Rate	
Ingestion	
Adult - Soil	100 mg/d
Child - Soil	200 mg/d
Adult - Groundwater	2 L/d
Inhalation	
Adult - Soil	20 m^3/d
Adult - Groundwater (volatiles)	15 m^3/d
Fish Ingestion[c]	54 g/d
Garden Produce[b]	
Root (e.g., carrots)	0.88 g/d
Leafy (e.g., lettuce)	1.1 g/d
Garden fruit (e.g., tomato)	2.2 g/d
Potato	9.1 g/d
Exposure Frequency	350 d/yr 2.6 h/d, 7 d/yr (swimming)
Exposure Duration	
Soil Ingestion and Dermal	
Adult	24 yr
Child	6 yr
All other pathways	30 yr
Body Weight	
Adult	70 kg
Child	15 kg
Averaging Time	
Carcinogens	70 yr x 365 d/yr
Non-carcinogens	30 yr x 365 d/yr
Skin Surface Area	
Adult - Soil	5000 cm^2 (summer); 1900 cm^2 (winter)
Child - Soil	3900 cm^2
Adult - Swimming	20,000 cm^2
Soil to Skin Adherence Factor	1 mg/cm^2/d
Contaminant-Specific Absorption Factor	
Inorganics[d]	0.001
BEHP[e]	0.0055
All other organics[d]	0.06
Permeability Coefficient - Trichloroethene[d]	4E-01 cm/hr
Groundwater Volatilization Factor[a]	0.5 L/m^3

[a]Factors based on EPA-10 (1991) unless otherwise specified
[b]EPA (1986a)
[c]EPA (1991a)
[d]EPA (1992c)
[e]Calculated factor; see Section 3.3.2

Dermal Exposure

The intake equation provided above is modified to provide the absorbed dose equation for dermal exposures to contaminated soil. Exposure factors, as provided in EPA-10 (EPA-10, 1991) are indicated.

$$\text{Dermally Absorbed Dose} = \frac{(CS \times CF \times ABS \times AF)\left[\left(\frac{SA \times EF \times ED}{BW}\right)_{child} + \left(\frac{SA \times EF \times ED}{BW}\right)_{adult}\right]}{AT} \quad (2)$$

where :

Dermally absorbed dose = (mg/kg-d)

CS	=	maximum concentration of contaminant in soil (mg/kg)
SA	=	skin surface area available for contact (child: 3,900 cm^2, Adult: 5,000 cm^2-summer, 1,900 cm^2-winter)
AF	=	soil-to-skin adherence factor (1 mg/cm^2/day)
ABS	=	contaminant-specific absorption factor (unitless)
EF	=	event frequency (child: 1 event/day, 350 d/yr; adult: 1 event/day 350 d/yr with 90 d as summer and 260 d as winter)
ED	=	exposure duration (6 yr) child (24 yr) adult
CF	=	conversion factor (1E-06 kg/mg)
BW	=	body weight (15 kg) child (70 kg) adult
AT	=	averaging time (noncarcinogenic effects: 365 d/yr x 30 yr; carcinogenic effects: 365 d/yr x 70 yr)

1.1 INTAKE CALCULATIONS

The following subsections present intake calculations for the soil ingestion, fugitive dust inhalation and dermal exposure pathways.

1.1.1 Soil Ingestion

Non-Carcinogenic

$$\text{Intake mg/kg-d} = \frac{(C \text{ mg/kg})(1E-06 \text{ kg/mg})\left[\left(\frac{(200 \text{ mg/d})(350 \text{ d/yr} \times 6 \text{ yr})}{15 \text{ kg}}\right)_{\text{child}} + \left(\frac{(100 \text{ mg/d})(350 \text{ d/yr} \times 24 \text{ yrs})}{70 \text{ kg}}\right)_{\text{adult}}\right]}{(365 \text{ d/yr} \times 30 \text{ yr})}$$

$$= C \text{ mg/kg} \times 3.7E-06 \text{ d}^{-1} \quad (3)$$

Carcinogenic

$$\text{Intake mg/kg-d} = \frac{(C \text{ mg/kg})(1E-06 \text{ kg/mg})\left[\left(\frac{(200 \text{ mg/d})(350 \text{ d/yr} \times 6 \text{ yr})}{15 \text{ kg}}\right)_{\text{child}} + \left(\frac{(100 \text{ mg/d})(350 \text{ d/yr} \times 24 \text{ yrs})}{70 \text{ kg}}\right)_{\text{adult}}\right]}{(365 \text{ d/yr} \times 70 \text{ yr})}$$

$$= C \text{ mg/kg} \times 1.6E-06 \text{ d}^{-1} \quad (4)$$

1.1.2 Inhalation

Intakes for the inhalation of fugitive dust are calculated for a residential receptor at each subunit and are based on fugitive dust emissions from that subunit only. Contaminant specific concentrations within fugitive, dust are calculated by multiplying the subunit specific dust concentration in table 3-1, with the maximum contaminant concentration in soil table 2-1.

Non-Carcinogenic

$$\text{Intake mg/kg-d} = \frac{(C \text{ mg/m}^3)(20 \text{ m}^3\text{/d})(350 \text{ d/yr})(30 \text{ yr})}{(70 \text{ kg})(30 \text{ yr} \times 365 \text{ d/yr})} = C \text{ mg/m}^3 \times 0.27 \text{ m}^3\text{/kg-d} \quad (5)$$

Carcinogenic

$$\text{Intake mg/kg-d} = \frac{(\text{C mg/m}^3)(20\ \text{m}^3/\text{d})(350\ \text{d/yr})(30\ \text{yr})}{(70\ \text{kg})(70\ \text{yr} \times 365\ \text{d/yr})} = \text{C mg/m}^3 \times 0.12\ \text{m}^3/\text{kg-d} \quad (6)$$

1.1.3 Dermal Absorption

Non-Carcinogenic

$$\text{Dermally Absorbed Dose mg/kg-d} =$$

$$\frac{(\text{CS mg/kg})(1\text{E-}06\ \text{kg/mg})(\text{ABS})(1\ \text{mg/cm}^2\text{-d})}{\left[\left\{\frac{(3900\ \text{cm}^2)(350\ \text{d/yr})(6\ \text{yr})}{15\ \text{kg}}\right\}\ \text{child} + \left\{\frac{(5000\ \text{cm}^2)(90\ \text{d/yr})(24\ \text{yr})}{70\ \text{kg}} + \frac{(1900\ \text{cm}^2)(260\ \text{d/yr})(24\ \text{yr})}{70\ \text{kg}}\right\}\ \text{adult}\right]}{(365\ \text{d/yr} \times 30\ \text{yr})}$$

$$= \text{CS mg/kg} \times \text{ABS} \times 7.9\text{E-}05\ \text{d}^{-1} \quad (7)$$

See table D-1 for ABS values (contaminant-specific absorption factors) and sources.

Carcinogenic

$$\text{Dermally Absorbed Dose mg/kg-d} =$$

$$\frac{(\text{CS mg/kg})(1\text{E-}06\ \text{kg/mg})(\text{ABS})(1\ \text{mg/cm}^2\text{-d})}{\left[\left\{\frac{(3900\ \text{cm}^2)(350\ \text{d/yr})(6\ \text{yr})}{15\ \text{kg}}\right\}\ \text{child} + \left\{\frac{(5000\ \text{cm}^2)(90\ \text{d/yr})(24\ \text{yr})}{70\ \text{kg}} + \frac{1900\ \text{cm}^2)(260\ \text{d/yr})(24\ \text{yr})}{70\ \text{kg}}\right\}\ \text{adult}\right]}{(365\ \text{d/yr} \times 70\ \text{yr})}$$

$$= \text{CS mg/kg} \times \text{ABS} \times 3.4\text{E-}05\ \text{d}^{-1} \quad (8)$$

See table III-1 for ABS values (contaminant-specific absorption factors) and sources.

1.2 EXAMPLE CALCULATIONS

All example intake calculations are made using the maximum contaminant concentrations for arsenic at the HRL. Calculations are not performed for the non-carcinogenic inhalation pathway because none of the COPC have an inhalation RfD.

1.2.1 Soil Ingestion

Non-Carcinogenic

$$6.6 \text{ mg/kg} \times 3.7\text{E-}06 \text{ d}^{-1} = 2.4\text{E-}05 \text{ mg/kg-d}$$

Carcinogenic

$$6.6 \text{ mg/kg} \times 1.6\text{E-}06 \text{d}^{-1} = 1.0\text{E-}05 \text{ mg/kg-d}$$

1.2.2 Inhalation

The concentration of arsenic in air, contributed to the residential receptor via the inhalation of fugitive dust from the HRL is:

$$C\ (mg/m^3) = U\ (mg/kg) \times D\ (\mu g/m^3) \times CF\ (kg/\mu g) \quad (9)$$

where:

C = Contaminant concentration of arsenic in air.
U = maximum contaminant concentration in soil for arsenic at the HRL (table 2-1).
D = Dust concentration at residential receptor for the HRL (table 3-1).
CF = Conversion Factor = 1E-09 kg/μg.

$$C = 6.6 \text{ mg/kg} \times 9.93\ \mu g/m^3 \times 1E{-}09\ kg/\mu g = 6.6E{-}08\ (mg/m^3) \quad (10)$$

Therefore,

Carcinogenic

$$\text{Intake} = 6.6\text{E-}8 \text{ mg/m}^3 \times 0.12 \text{ m}^3\text{/kg-d} \times .30^{*} = 2.4\text{E-}09 \text{ (mg/kg-d)}$$

*Assumes approximately 30 percent of the inhaled dose of arsenic is absorbed

Non-Carcinogenic

Not applicable.

1.2.3 Dermal Absorption

Non-Carcinogenic

$$6.6 \text{ mg/kg} \times .001 \times 7.9\text{E-}05 \text{ d}^{-1} = 5.2\text{E-}07 \text{ mg/kg-d}$$

Carcinogenic

$$6.6 \text{ mg/kg} \times .001 \times 3.4\text{E-}05 \text{ d}^{-1} = 2.2\text{E-}07 \text{ mg/kg-d}$$

2.0 CALCULATION OF CONTAMINANT INTAKES FOR THE GARDEN PATHWAY

Calculation of contaminant intakes was performed for 4 categories of vegetables:

1) Leafy (lettuce)
2) Root (carrot)
3) Garden vegetable (tomato)
4) Potato

2.1 PLANT CONCENTRATIONS

Before intakes can be calculated a contaminant concentration within each plant must be determined via the following equation:

$$CP = SC \times UF$$

where:

CP = concentration in plant mg/kg
SC = maximum soil concentration mg/kg
UF = uptake factor (unitless)

Table III-2 presents the uptake factors specific to each vegetable category.

Table III-2. Summary of Plant Uptake Factors[a,b].

Contaminant	Leafy	Root	Garden Fruits	Potatoes
Arsenic	0.04	0.02	0.002	0.0006
BEHP[c]	0.38	0.36	0.02	0.02
Beryllium[d]	0.43	0.26	0.041	0.06
Chlordane	0.02[e]	2.02[f]	0.21[e]	0.3[e]
Chromium	0.2[g]	0.26[d]	0.041[d]	0.06[d]
PCBs	0.38	0.36	0.02	0.02
Tetrachloroethene	NA	NA	NA	NA
1,1,1-Tetrachloroethane	NA	NA	NA	NA
Trichloroethene	NA	NA	NA	NA

[a]All uptake factors expressed as [μg/g tissue DW (μg/g soil)$^{-1}$]
[b]Source: EPA 1986a unless otherwise indicated
[c]PCB uptake factors used as surrogates for BEHP
[d]95% UCL of mean for uptake factors of As, Cd, Pb, Hg, Ni, Se, Zn (EPA 1986a)
[e]Heptachlor uptake factors used as surrogates for chlordane
[f]95% UCL of mean for uptake of chlordane by sugar beets
[g] Kabata - Pendias and Pendias 1984

NA Indicates not applicable

2.1.1 Calculation of Contaminant Concentration in the Four Vegetable Categories

All example calculations use the soil concentration of arsenic at HRL.

Leafy (Lettuce)

$$CP\ mg/kg = 6.6\ mg/kg \times 0.04 = 0.26\ mg/kg$$

Root (Carrots)

$$CP\ mg/kg = 6.6\ mg/kg \times 0.02 = 0.13\ mg/kg$$

Garden Vegetable (tomato)

$$CP\ mg/kg = 6.6\ mg/kg \times 0.002 = 0.013\ mg/kg$$

Potato

$$CP\ mg/kg = 6.6\ mg/kg \times 0.0006 = 0.004\ mg/kg$$

2.2 INTAKE CALCULATIONS

The following section presents intake calculations for the four vegetable groups (leafy, root, garden vegetable, and potato).

The basic intake equation is:

$$\text{Intake mg/kg-d} = \frac{CP \times IR \times EF \times ED \times CF}{BW \times AT} \quad (11)$$

where:

CP	=	concentration in plant mg/kg
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
CF	=	conversion factor (1E-03) kg/g
BW	=	body weight (70 kg)
AT	=	averaging time: carcinogens (365 d/yr x 70 yrs) non-carcinogens (365 d/yr x 30 yrs)
IR	=	intake rate for specific vegetable (g/d)

Vegetable Group	Intake Rate (g/d)
Leafy (lettuce)	1.1
Root (carrot)	0.88
Garden vegetable (tomato)	2.2
Potato	9.1

Non-Carcinogenic

$$\text{Intake mg/kg-d} = \frac{\text{(CP mg/kg)(IR g/d)(350 d/yr)(30 yr)(1E-03 kg/g)}}{\text{(70 kg)(365 d/yr x 30 yr)}} \quad (12)$$

$$\text{Intake mg/kg-d} = \text{CP mg/kg x IR g/d x 1.4E-05 g}^{-1}$$

Carcinogenic

$$\text{Intake mg/kg-d} = \frac{\text{(CP mg/kg)(IR g/d)(350 d/yr)(30 yr)(1E-03 kg/g)}}{\text{(70 kg)(365 d/yr x 70 yr)}} \quad (13)$$

$$\text{Intake mg/kg-d} = \text{CP mg/kg x IR g/d x 5.9E-06 g}^{-1}$$

2.3 EXAMPLE CALCULATIONS

Example calculations for the noncarcinogenic intakes are made using concentrations for arsenic at the HRL. As discussed in section 4.2, arsenic in plants is predominatly in organic forms that are not carcinogenic. Therefore, beryllium is used to calculate the example carcinogenic intake.

Non-Carcinogenic (leafy) - arsenic

$$\text{Intake} = 0.26 \text{ mg/kg x } 1.1 \text{ g/d x } 1.4\text{E-}05 \text{ g}^{-1} = 4\text{E-}06 \text{ mg/kg-d}$$

Carcinogenic (leafy) - beryllium

$$\text{Intake} = 0.56 \text{ mg/kg x } 1.1 \text{ g/d x } 5.9\text{E-}06\text{g}^{-1} = 3.6\text{E-}06 \text{ mg/kg-d}$$

The additional three vegetable categories are calculated in the same manner with the group specific intake rate (see section 3.2) and plant contaminant concentrations (table 3-3) as the two variables.

3.0 CALCULATION OF CONTAMINANT INTAKES FOR THE GROUNDWATER PATHWAYS

As in sections D2.0 and D3.0, Standard EPA Equations for calculation of contaminant intakes, as provided in RAGS (EPA, 1989a) and EPA (1991a) are used as the basis for groundwater contaminant intake calculations.

The basic equation for calculating intakes via groundwater ingestion or volatile inhalation is:

$$\text{Intake} = \frac{C \times IR \times EF \times ED}{BW \times AT} \quad (14)$$

where:

Intake	=	estimated contaminant intake (mg/kg-d)
C	=	estimated water concentration (mg/L)
IR	=	contact rate (2 L/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time: carcinogens (365 d/yr x 70 yrs) non-carcinogens (365 d/yr x 30 yrs)

For volatile inhalation the equation is modified to include a volatilization factor (K):

Therefore,

$$\text{Intake} = \frac{CW \times IR \times EF \times ED}{BW \times AT} \quad (15)$$

where:

Intake	=	estimated contaminant intake (mg/kg-d)
CW	=	estimated water concentration (mg/L) x K volatilization factor (0.5 L/m^3)
IR	=	contact rate (15 L/d)
EF	=	exposure frequency (350 d/yr)
ED	=	exposure duration (30 yr)
BW	=	body weight (70 kg)
AT	=	averaging time: carcinogens (365 d/yr x 70 yrs) non-carcinogens (365 d/yr x 30 yrs)

3.1 Intake Calculations

The following Subsections present intake calculations for the groundwater ingestion and volatile inhalation pathways.

3.1.1 Groundwater Ingestion

Non-Carcinogenic

$$\text{Intake mg/kg-d} = \frac{\text{(C mg/L)(2 L/d)(350 d/yr)(30 yr)}}{\text{(70 kg)(365 d/yr)(30 yr)}} \quad (16)$$

$$= \text{C mg/L x 0.027 L/kg-d}$$

Carcinogenic

$$\text{Intake mg/kg-d} = \frac{\text{(C mg/L)(2 L/d)(350 d/yr)(30 yr)}}{\text{(70 kg)(365 d/yr x 70 yr)}} \quad (17)$$

$$= \text{C mg/L x 0.012 L/kg-d}$$

3.1.2 Inhalation of Volatiles

Non-Carcinogenic

Not applicable.

Carcinogenic

$$\text{Intake mg/kg-d} = \frac{(C \text{ mg/L})(15 \text{ m}^3\text{/d})(350 \text{ d/yr})(30 \text{ yr})(0.54 \text{ m}^3)}{(70 \text{ kg})(365 \text{ d/yr} \times 70 \text{ yr})} \quad (18)$$

$$= C \text{ mg/L} \times 4.4E\text{-}02 \text{ L/kg-d}$$

3.2 EXAMPLE CALCULATIONS

Example calculations are performed using the maximum contaminant concentrations for nitrate and trichloroethene as appropriate.

3.2.1 Groundwater Ingestion

Non-Carcinogenic - Nitrate

$$61 \text{ mg/L} \times 0.027 \text{ L/kg-d} = 1.7 \text{ mg/kg-d} \quad (19)$$

Carcinogenic - Trichloroethene

$$0.11 \text{ mg/L} \times 0.012 \text{ L/kg-d} = 1.3E\text{-}03 \text{ mg/kg-d} \quad (20)$$

3.2.2 Inhalation of Volatiles

Non-Carcinogenic

Not applicable.

Carcinogenic - Trichloroethene

$$0.11\ mg/L \times 4.4E{-}02\ L/kg{-}d = 4.8E{-}03\ mg/kg{-}d \quad (21)$$

4.0 CALCULATION OF HUMAN HEALTH ASSESSMENT

Hazard Quotient

The basic equation for determining the HQ for all pathways is:

$$HQ = I/RfD$$

where:

HQ	=	hazard quotient (unitless)
I	=	intake (mg/kg-d)
RfD	=	contaminant-specific chronic reference dose (mg/kg-d)

Incremental Cancer Risk

The basic equation for determining the ICR for all pathways is:

$$ICR = I \times SF$$

where:

ICR	=	lifetime incremental cancer risk (unitless)
I	=	intake (mg/kg-d)
SF	=	contaminant-specific slope factor $(mg/kg\text{-}d)^{-1}$

Note: All ICR calculations are made to one significant figure only.

4.1 EXAMPLE CALCULATIONS

All example calculations are made using values for arsenic at the HRL with the exception of the HQ for the Inhalation Pathway. No HQ's have been calculated for this pathway since there are no published inhalation RfD's available for any of the COPC.

4.1.1 Soil Pathway

4.1.1.1 Soil Ingestion

Hazard Quotient

$$HQ = \frac{2.4E-05\ mg/kg-d}{3.0E-04\ mg/kg-d} = 0.08 \quad (22)$$

Incremental Cancer Risk

$$ICR = (1.0E-05\ mg/kg-d \times 1.7\ (mg/kg-d)^{-1} = 2E-05 \quad (23)$$

4.1.1.2 Inhalation of Fugitive Dust

Hazard Quotient - Not Applicable

Incremental Cancer Risk

$$ICR = 2.4E\text{-}09\ mg/kg\text{-}d \times 50\ (mg/kg\text{-}d)^{-1} = 1E\text{-}07^{*}$$

*The slope factor for arsenic is based on 30 percent absorption of the inhaled arsenic. Therefore, intakes have been adjusted accordingly for arsenic, to determine the ICR.

4.1.1.3 Dermal Exposure

Hazard Quotient

$$HQ = \frac{5.2E-07\ mg/kg-d}{3.0E-04\ mg/kg-d} = 0.002 \quad (24)$$

Incremental Cancer Risk

$$ICR = 2.2E\text{-}07\ mg/kg\text{-}d \times 1.7\ (mg/kg\text{-}d)^{-1} = 4E\text{-}07$$

4.1.2 Garden Pathway

The values used to calculate HQ and ICR for the garden pathway are the total contaminant intake, *i.e.*, the sum of all the intakes for arsenic for the four vegetable groups combined. As discussed in section 4.2, arsenic in plants is predominantly in organic forms that are not carcinogenic. Therefore, beryllium is used for the example ICR calculation.

Hazard Quotient - arsenic

$$HQ = \frac{6.4E-06 \text{ mg/kg-d}}{3.0E-04 \text{ mg/kg-d}} = 0.02 \quad (25)$$

Incremental Cancer Risk - beryllium

$$ICR = 1E\text{-}05 \text{ mg/kg-d} \times 4.3 \text{ (mg/kg-d)}^{-1} = 4E\text{-}05$$

4.1.3 Groundwater Pathway

4.1.3.1 Groundwater Ingestion

Hazard Quotient - nitrate

$$HQ = \frac{1.7 \text{ mg/kg-d}}{1.6 \text{ mg/kg-d}} = 1 \quad (26)$$

Incremental Cancer Risk - trichloroethene

$$ICR = 1.3E-03 \text{ mg/kg-d} \times 1.1E-02 \text{ (mg/kg-d)}^{-1} = 1E-05 \quad (27)$$

4.1.3.2 Inhalation of Volatiles

Hazard Quotient

Not applicable.

Incremental Cancer Risk - trichloroethene

$$ICR = 4.8E-03 \text{ mg/kg-d} \times 6.0E-03 \text{ (mg/kg-d)}^{-1} = 3E-05 \quad (28)$$

APPENDIX IV

STATISTICAL INFORMATION FOR BISRA AND BRSRA

This page left intentionally blank.

This appendix presents the methodologies and results for the calculation of the 95 percent upper confidence limit (UCL) of the mean contaminant concentration. Soil contaminants are discussed in section 1.0 and groundwater contaminants are discussed in section 2.0. A discussion of upper tolerance limit calculations is provided in section 3.0.

1.0 CALCULATION OF 95 PERCENT UCL'S FOR SOIL CONTAMINANTS

To calculate the 95 percent UCL, data were used that approximately represented the distribution of specific contaminants for each site. Data that were rejected by validation were not included in calculations. All data from the Phase I and Phase II RI's were considered but not all data were used in the calculations. Selected data at the Horn Rapids Landfill (HRL) and the UN-1100-6 subunit were selected to provide analyses of "hot spots" for soil and the contaminant plume in the groundwater in the vicinity of the HRL, as discussed below. This provides a conservative bias to the 95 percent UCL for certain contaminants. For a contaminant of concern, specific to a subunit, one-half the sample quantitation limit (SQL) (DOE-RL 1992) was used in the calculations when a contaminant of concern was not detected in a sample. These are reported at one-half the SQL (*i.e.*, noted with a U qualifier) in all tables in this section. Anywhere PCB's were detected, the measured concentrations or one-half the SQL, were summed for all the Arochlors detected at that subunit.

Phase I soil data used in the calculations were taken from DOE-RL (1990) and Phase II soil data is presented in appendix D.

95 percent UCL was calculated as follows (Hines and Montgomery, 1980):

95% UCL	=	Sample average + $t_{\alpha, df}$ (sample standard deviation/square root (n))
n	=	sample size
t	=	Student's t statistic for α, df (*i.e.*; degrees of freedom)

where:

α	=	0.05
df	=	n-1

The 95 percent UCL's for soil contaminants are summarized in table V-1. The data used for calculating the UCL's is provided in tables V-2, V-3, and V-4 for the UN-1100-6 subunit, the Ephemeral Pool, and HRL, respectively.

Table IV-1. Summary of Statistical Calculation Information for Soils.

Location	Contaminant	Sample Mean Concentration mg/kg	Sample Standard Deviation mg/kg	Sample Number	95% UCL mg/kg
Ephemeral Pool	Chlordane	1.4	0.89	9	1.9
Ephemeral Pool	Total PCBs	6.5	14	9	15
UN-1100-6	BEHP	13,000	6,400	6	18,000
UN-1100-6	Chlordane	1.1	0.56	6	1.6
Horn Rapids Landfill	Arsenic	1.3	0.7	100	1.4
Horn Rapids Landfill	Beryllium	0.5	0.3	100	0.5
Horn Rapids Landfill	Chromium	44	170	55	83
Horn Rapids Landfill	Total PCBs	28	26	22	38

Table IV-2. Summary of Phase I BEHP and Chlordane Surface Soil Sampling at UN-1100-6.

Sample No.	BEHP ug/kg	Q	*Chlordane ug/kg	Q
S6150	25000000		1860	J
S6151	6700000		590	J
S6152	8900000		1780	J
S6153	11000000		820	J
S6154	13000000		960	J
S6155	14000000		670	J

BEHP - Bis(2-ethylhexyl)phthalate
* Chlordane is sum of alpha and gamma chlorane
Q - data qualifier

Table IV-3. Summary of Phase 1 and Phase 2 Soil Sampling Data at the Ephemeral Pool.

SDG	Boring Loc.	Sample No.	Sample Depth (ft)	Total PCB's ug/kg	Q	*Chlordane ug/kg	Q
PHASE I DATA							
S6150A	UNK	S6164A	0-0.5	4700		480	
	UNK	S6165A	0-0.5	300	J	1810	
PHASE II DATA							
B00G51	E1	B00G76	S	170	U	2800	
	E2	B00G51	S	42000		950	
	E3	B00G52	S	11000	J	700	
	E4	B00G53	S	165	U	540	
	E4	B00G54	S	170	U	730	
	E5	B00G77	S	175	U	2560	
	E6	B00G56	S	190	U	1710	

PCB's - polychlorinated biphenyls
* Chlordane is sum of alpha and gamma chlorane
Q - data qualifier

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 1 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
PHASE I DATA								NA		NA	
AH168S/ A1307S		AH168S	0-0.5	0.65	J	0.46		NA		NA	
		AH169S	0-0.5	1.5	J	0.09	U	NA		NA	
		AH171S	0-0.5	2.1	J	0.42		NA		NA	
		AH172S	0-0.5	1.9		0.79		NA		NA	
		AH173S	0-0.5	0.67	J	0.105	U	NA		NA	
		AH174S	0-0.5	1.1	J	0.08	U	NA		NA	
		AH175S	0-0.5	1.6		0.08	U	NA		NA	
		AH176S	0-0.5	1.1		0.085	U	NA		NA	
		AH177S	0-0.5	1.7		0.22		NA		NA	
		AH178S	0-0.5	0.96	J	0.2		NA		NA	
		AH179S	0-0.5	1	J	0.085	U	NA		NA	
AH180S/ A1312S		AH180S	0-0.5	0.62		0.085	U	NA		NA	
		AH181S	0-0.5	2.3		0.83		NA		NA	
		AH184S	0-0.5	0.87		0.13		NA		NA	
		AH185S	0-0.5	3.6		0.67		NA		NA	
AH186S		AH186S	0-0.5	1.1		0.09	U	NA		NA	
AH186S		AH187S	0-0.5	1.3		0.085	U	NA		NA	
		AH188S	0-0.5	1.1		0.09	U	NA		NA	

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 2 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
		AH189S	0-0.5	1.8		0.095	U	NA		NA	
		AH190S	0-0.5	2.1		0.18	U	NA		NA	
		AH191S	0-0.5	1.4		0.08	U	NA		NA	
		AH192S	0-0.5	1.5		0.08	U	NA		NA	
		AH193S	0-0.5	1.2		0.09	U	NA		NA	
		AH194S	0-0.5	1.1		0.095	U	NA		NA	
		AH195S	0-0.5	1.8		0.095	U	NA		NA	
		AH196S	0-0.5	1.8		0.085	U	NA		NA	
		AH197S	0-0.5	1.7		0.085	U	NA		NA	
		AH198S	0-0.5	2.2		0.09	U	NA		NA	
		AH199S	0-0.5	1.3		0.085	U	NA		NA	
		AH200S	0-0.5	1.5		0.08	U	NA		NA	
		AH201S	0-0.5	0.92		0.07	U	NA		NA	
		AH202S	0-0.5	1.9		0.08	U	NA		NA	
		AH203S	0-0.5	0.71		0.07	U	NA		5000	J
		AH204S	0-0.5	1.9		0.08	U	NA		NA	
		AH205S	0-0.5	1.8		0.09	U	NA		NA	

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 3 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
AH206S		AH206S	0-0.5	1.9		0.62		NA		NA	
		AH207S	0-0.5	1.2	J	1.1		NA		NA	
		AH208S	0-0.5	1.6	J	1		NA		NA	
		AH209S	0-0.5	1.2	J	0.94		NA		NA	
		AH211S	0-0.5	1.9	J	0.85		NA		NA	
		AH212S	0-0.5	1.8	J	0.98		NA		NA	
		AH213S	0-0.5	1.4	J	1		NA		NA	
		AH214S	0-0.5	2.1	J	0.52		NA		NA	
		AH215S	0-0.5	NR		NR		NA		NA	
A1615S	HRL-2	A1802S	0-2.5	1.2		0.42		9		NA	
		A1804S	5.1-7.9	1.3	J	0.52		6.6	J	NA	
		A1805S	5.1-7.9	1.1	J	0.55		6	J	NA	
		A1807S	13.9-16.2	0.67	J	0.57		5.1	J	NA	
		A1810S	13.9-16.2	0.67	J	0.55		7.3	J	NA	
A1901S	HRL-3	A2002S	0-2.5	2.2		0.59		13.2		NA	
		A2004S	4.6-7.5	1.3		0.56		7.6	J	NA	
		A2005S	4.6-7.5	1.8		0.69		6.6	J	NA	
		A2007S	10.8-13	1.4	J	0.62		4.6		NA	
A1901S	HRL-3	A2009S	14.5-17	1.4		0.78		7	J	NA	

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 4 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
A1912S	HRL-4	A2202S	0-2.8	0.82	J	0.85		4.1		65000	J
		A2204S	5.4-8	1.5		0.97		7.4		NA	
		A2205S	5.4-8	1.1		0.87		6.2		NA	
		A2207S	10.5-13.6	1		1.1		10		NA	
		A2209S	14.6-16.9	1.7		1.1		1250		NA	
A1501W	HRL-5	A1502S	0-2.1	1.1	J	0.58		5.7	J	NA	
		A1503S	3.8-6	0.56	J	0.54		4.1	J	NA	
		A1504S	0.4-8.6	0.71	J	0.71		5.2	J	NA	
		A1506S	9.4-11.6	0.79	J	0.8		6.1	J	NA	
		A1507S	9.4-11.6	0.79	J	0.66		6.2	J	NA	
		A1509S	13.1-15.5	0.76	J	0.73		81.5	J	NA	
	HRL-6	A1601S	2.4-4.8	0.67	J	0.38		7.9	J	NA	
		A1602S	4.8-7.1	0.81	J	0.58		7.8	J	NA	
		A1604S	7.1-9.4	0.72	J	0.48		4.8	J	NA	
		A1606S	9.4-11.6	0.91	J	0.33		5.8	J	NA	
		A1607S	11.6-13.9	0.57	J	0.59		13.7	J	NA	
		A1608S	11.6-13.9	0.72	J	0.52		8	J	NA	
A2214S	HRL-7	A2301S	0-2.5	1.3	J	0.69		8.8		NA	
		A2303S	4.8-7.2	0.94	J	0.28		7.6		NA	

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 5 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
A2214S	HRL-7	A2304S	4.8-7.2	0.82	J	0.54		9.7		NA	
		A2306S	8.9-11.2	4.2	J	0.76		6.5		NA	
		A2310S	12.7-15.1	0.97	J	0.61		9.1		NA	
A1401W	HRL-8	A1402S	0-2.5	1		0.95		16.2		NA	
		A1404S	5.9-7.4	0.73		0.73		11.4		NA	
		A1406S	8.7-10.9	0.2		1		284		NA	
		A1408S	10.9-12.8	0.45		0.89		72		NA	
		A1409S	15-17.3	1.1		1		119		NA	
A1615S	HRL-9	A1701S	0-2.5	0.76	J	0.44		5	J	NA	
		A1704S	3.7-4.6	0.46	J	0.51		24.9	J	NA	
		A1706S	6.8-9.1	0.58	J	0.62		14	J	NA	
		A1707S	6.8-9.1	0.37	J	0.48		13.2	J	NA	
		A1709S	10.9-13.1	0.48	J	0.42		4.7	J	NA	
A1901S	HRL-10	A1901S	0-2.3	1.9		0.37		10.8	J	NA	
		A1902S	2.3-4	1.7		0.61		17.6	J	NA	
		A1905S	6.9-9.1	1.5		0.69		9.9	J	NA	
		A1906S	6.9-9.1	1.8		0.6		9.6	J	NA	

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 6 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
PHASE II DATA											
WHC 23	TP-11	B00Z59	4	4.1		0.115	U	85.7		NA	
WHC 28	TP-3B	B00ZT3	7-7.5	R		R		4.9	J	NA	
	TP-3B	B00ZT4	7-7.5	R		R		4.3	J	NA	
	TP-3A	B00ZT7	5	R		R		3.7	J	NA	
	TP-3A	B00ZT8	10	R		R		9.9	J	NA	
	TP-4/5	B00ZV1	5	R		R		3.2	J	NA	
	TP-4/5	B00ZV2	12	R		R		133	J	NA	
WHC 29	TP-8	B00ZV3	5	0.74	B	0.55	B	19.8		NA	
WHC 27	TP-7	B00ZT2	5	2.9	J	0.115	U	9.8		NA	
WHC 23	TP-1	B00ZT0	5	NA		NA		NA		NA	
	TP-1	B00ZT1	9	NA		NA		NA		NA	
WHC 30	B5-2	B00ZX5	1	NA		NA		NA		NA	
WHC 31	B5-3	B00ZX7	S	NA		NA		NA		NA	
	B5-3	B00ZY0	1'	NA		NA		NA		NA	
WHC 30	B4-1	B00ZW6	S	NA		NA		NA		NA	
	B4-1	B00ZW7	1	NA		NA		NA		NA	
WHC 31	B5-3	B00ZX9	S	NA		NA		NA		NA	
WHC 6	B5-3	B00GB0	0-1	1.2	J	0.55	B	NA		NA	
WHC 6	B5-3	B00GB1	1-2	1.2	J	0.48	B	NA		NA	

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 7 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
	B5-2	B00GB2	0-1	0.86	J	0.42	B	NA		NA	
	B5-2	B00GB3	1-2	0.76	J	0.42	B	NA		NA	
	B4-1	B00GB4	0-1	1.8	J	1	B	NA		NA	
	B4-1	B00GB5	0-1	1.8	J	1.1	B	NA		NA	
	B4-1	B00GB7	1-2	1.2	J	0.77	B	NA		NA	
	PCB-1	B00G92	0-1	NA		NA		NA		49000	J
	PCB-1	B00G93	1-2	NA		NA		NA		41000	J
	PCB-2	B00G94	0-1	NA		NA		NA		80000	J
	PCB-2	B00G95	1-2	NA		NA		NA		100,000	J
	PCB-3	B00G96	0-1	NA		NA		NA		6100	J
	PCB-3	B00G97	1-2	NA		NA		NA		15000	J
	PCB-4	B00G98	0-1	NA		NA		NA		21000	J
	PCB-4	B00G99	1-2	NA		NA		NA		1500	J
WHC 30	PCB-2A	B00ZV4	1	NA		NA		NA		8500	B
	PCB-2A	B00ZV5	1.5	NA		NA		NA		12000	B
	PCB-3A	B00ZV6	S	NA		NA		NA		3500	B
	PCB-3A	B00ZV7	1	NA		NA		NA		23000	B
	PCB-3A	B00ZV8	20"	NA		NA		NA		9700	B
	PCB-4A	B00ZV9	S	NA		NA		NA		16000	B
WHC 30	PCB-2A	B00ZX6	1.5	NA		NA		NA		2300	B

Table IV-4. Summary of Phase I and Phase II Soil Sampling at the Horn Rapids Landfill (sheet 8 of 8).

SDG	Boring Loc.	Sample No.	Sample Depth	Arsenic mg/kg	Q	Beryllium mg/kg	Q	Chromium mg/kg	Q	Total PCBs ug/kg	Q
	PCB-4A	B00ZW1	S	NA		NA		NA		36000	B
	PCB-4A	B00ZW2	1	NA		NA		NA		39000	B
	PCB-1A	B00ZW3	S	NA		NA		NA		20000	B
	PCB-1A	B00ZW4	1	NA		NA		NA		29000	B
	PCB-1A	B00ZW5	1.5	NA		NA		NA		43000	B

PCB's - polychlorinated biphenyls
Q - data qualifier

Table IV-5. Summary of Statistical Calculation Information for Groundwater at Horn Rapids Landfill.

Contaminant, units	Sample Mean	Sample Standard Deviation	95% UCL of Mean Conc.	Sample Number
TCE, mg/L	71	13	75	39
NO3-N, mg/L	43	8	45	58
Alpha, pCi/L	4.3	3	5	49
Beta, pCi/L	60	21	65	53

TCE - Trichloroethene
UCL - Upper confidence limit

This page left intentionally blank.

Table IV-6. Summary of Groundwater Sampling Data (Non-Radioactive) at Horn Rapids Landfill. (sheet 1 of 2)

Well	Round	Trichloroethene (mg/L)	Nitrate as nitrogen (mg/L)
MW-10	1	--	38.4
	2	--	36.9
	3	--	42.1
	4	--	38.3
	5	--	39
	6	--	38
	7	--	47
	7.5	--	38
	8	--	42
	9	--	43
MW-11	1	--	40.6
	2	--	40.5
	3	--	47.8
	4	--	46.5
	5	--	40
	6	--	46
	7	--	39
	7.5	--	48
	8	--	NA
	9	--	49
MW-12	1	92	49
	2	110	49
	3	80	56.7
	4	74	50.8
	5	79	50
	6	78	49
	7	69	51
	7.5	67	52
	8	69	NA
	9	58	52
MW-13	1	90	47
	2	91	44.9
	3	81	60.6
	4	69	46.7
	5	68	45
	6	70	46
	7	69	45
	7.5	66	43
	8	63	NA

Table IV-6. Summary of Groundwater Sampling Data (Non-Radioactive) at Horn Rapids Landfill. (sheet 2 of 2)

Well	Round	Trichloroethene (mg/L)	Nitrate as nitrogen (mg/L)
MW-14	1	40	48.5
	2	73	50.9
	3	60	61
	4	66	49.9
	5	82	47
	6	75	47
	7	75	47
	7.5	76	48
	8	67	NA
	9	58	51
MW-15	1	84	32.3
	2	80	32.2
	3	82	44.3
	4	59	31
	5	60	30
	6	62	33
	7	70	30
	7.5	66	36
	8	64	NA
	9	34	24
MW-20	6	--	NA
	7	--	31
	7.5	--	31
	8	--	28
	9	--	35

-- Data not used in statistical calculations
NA Not available

Table IV-7. Summary of Groundwater Sampling Data (Radioactive) at Horn Rapids Landfill. (sheet 1 of 2)

Well	Round	Alpha (pCi/L)	Beta (pCi/L)
MW-10	1	11.9	30.2
	2	<2.2	85.2
	3	<0	95.4
	4	6.6	88.9
	5	<2	63
	6	<3	62
	7	<1	18
	7.5	2.9	43
	8	<2	48
	9	NA	NA
MW-11	1	12.2	35.2
	2	<2.4	86.5
	3	6.6	74.7
	4	4.2	81
	5	<2	60
	6	<3	61
	7	<2	20
	7.5	<2	49
	8	9.6	60
	9	NA	NA
MW-12	1	7.6	34.6
	2	4.8	87.6
	3	NA	91
	4	6.5	77.6
	5	<2	61
	6	5.5	66
	7	NA	NA
	7.5	3.6	53
	8	<2	58
	9	NA	NA
MW-13	1	9.1	28.8
	2	4.1	71
	3	6.5	81.2
	4	5.8	85.8
	5	6.4	61
	6	<5	48
	7	NA	NA
	7.5	3.5	48
	8	2.9	51

Table IV-7. Summary of Groundwater Sampling Data (Radioactive) at Horn Rapids Landfill. (sheet 2 of 2)

Well	Round	Alpha (pCi/L)	Beta (pCi/L)
MW-14	1	6.3	25.1
	2	4.9	89.4
	3	9.6	90.8
	4	9.2	89
	5	< 3	70
	6	8.4	61
	7	NA	NA
	7.5	< 2	46
	8	5.3	56
	9	NA	NA
MW-15	1	9.3	23.2
	2	< 1.6	51.4
	3	3.7	63.6
	4	5	57.6
	5	< 2	46
	6	< 5	50
	7	NA	NA
	7.5	2.2	41
	8	3.5	43
	9	NA	NA
MW-20	6	--	NA
	7	--	71
	7.5	--	53
	8	--	87
	9	--	NA

-- Data not used in statistical calculations
NA Not available

Table IV-8. TAL Parameter UTLs for Background Soils (mg/kg). (Sheet 1 of 2)

Parameter	Operable Unit Specific Background									
	0-2 ft deep					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
aluminum	6703	943	8	8	9,710	4,270	898	11	11	6,236
antimony			8	0	3.70*			11	0	3.1*
arsenic	1.51	0.78	8	8	3.99	1.0	0.67	11	10	2.92**
barium	73.5	14.8	8	8	120	90.6	51.5	11	11	236
beryllium	0.32	0.13	8	7	0.74**	0.11	0.06	11	2	0.27**
cadmium	0.24	0.15	8	2	0.70**			11	0	0.36*
calcium	3073	645.2	8	8	5,130	5,443	848	11	11	7,830
chromium	9.19	1.18	8	8	12.9	13.5	12.01	11	11	47.3
cobalt	10.0	2.42	8	8	17.7	12.8	1.44	11	11	16.8
copper	11.1	2.50	8	8	19.1	16.09	1.22	11	11	19.5
iron	18,225	3,728	8	8	31,110	22,445	2,480	11	11	29,400
lead	5.04	2.38	8	8	12.6	2.8	0.95	11	11	5.0
magnesium	3,984	797	8	8	6,524	3,873	286	11	11	4,680
manganese	323	72.0	8	8	552	290	23.1	11	11	355
mercury			8	0	0.10*			11	0	0.1*
nickel	8.92	3.16	8	7	19.0**	10.8	35.4	11	11	26.0
potassium	1,318	186	8	8	1,910	643	115	11	11	966
selenium			8	0	0.39*			11	0	0.41*
silver	0.85	0.50	8	6	2.44**			21	0	0.54*

Table IV-8. TAL Parameter UTLs for Background Soils (mg/kg). (Sheet 2 of 2)

Parameter	Operable Unit Specific Background									
	0-2 ft deep					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
sodium	103	43.5	8	3	242**	306	40.4	11	11	419
thallium			8	0	0.39*			11	0	0.41*
vanadium	44.4	12.4	8	8	83.9	70.4	15.6	11	11	115
zinc	38.9	7.30	8	8	62.2	41.1	3.33	11	11	50.4
cyanide			8	0	0.52*			11	0	0.51*

$\bar{X}$ = Mean.
s = standard deviation.
n = number of samples.
d = number of detects.
UTL = upper 95 percentile tolerance limit.
*Parameter was never detected in the respective background samples; therefore, the highest reported respective background SQL is substituted as a surrogate UTL.
**Some non-detects present, 1/2 SQL used as surrogate value for corresponding sample.
[1]Does not include saturated soils.

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 1 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
Volatiles										
chloromethane			9	0	11			11	0	11
bromomethane			9	0	11			11	0	11
vinyl chloride			9	0	11			11	0	11
chloroethane			9	0	5			11	0	11
methylene chloride			9	0	5			11	0	5
acetone			9	0	43			11	0	22
carbon disulfide			9	0	5			11	0	5
1,1-dichloroethene			9	0	5			11	0	5
1,1-dichloroethane			9	0	5			11	0	5
1,2-dichloroethene			9	0	5			11	0	5
chloroform			9	0	5			11	0	5
1,2-dichloroethane			9	0	11			11	0	5
2-butanone			9	0	5			11	0	11
1,1,1-trichloroethane			9	0	5			11	0	5
carbon tetrachloride			9	0	11			11	0	5
vinyl acetate			9	0	5			11	0	11
bromodichloromethane			9	0	5			11	0	5
1,2-dichloropropane			9	0	5			11	0	5
cis-1,3-dichloropropene			9	0	5			11	0	5
trichloroethene			9	0	5			11	0	5

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 2 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
dibromochloromethane			9	0	5			11	0	5
1,1,2-trichloroethane			9	0	5			11	0	5
benzene			9	0	5			11	0	5
trans-1,3-dichloropropene			9	0	5			11	0	5
bromoform			9	0	5			11	0	5
4-methyl-2-pentanone			9	0	11			11	0	11
2-hexanone			9	0	11			11	0	11
tetrachloroethene			9	0	5			11	0	5
1,1,2,2-tetrachloroethane			9	0	5			11	0	5
toluene			9	0	5			11	0	5
chlorobenzene			9	0	5			11	0	5
ethylbenzene			9	0	5			11	0	5
styrene			9	0	5			11	0	5
xylene(total)			9	0	5			11	0	5
Semivolatiles										
phenol			9	1	38,100			11	0	350
bis(2-chloroethyl)ether			9	0	690			11	0	350
2-chlorophenol			9	0	690			11	0	350
1,3-dichlorobenzene			9	0	690			11	0	350
1,4-dichlorobenzene			9	0	690			11	0	350
benzyl alcohol			9	0	690			11	0	350

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 3 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
1,2-dichlorobenzene			9	0	690			11	0	350
2-methylphenol			9	0	690			11	0	350
bis(2-chloroisopropyl)ether			9	0	690			11	0	350
4-methylphenol			9	0	690			11	0	350
N-nitroso-di-n-propylamine			9	0	690			11	0	350
hexachloroethane			9	0	690			11	0	350
nitrobenzene			9	0	690			11	0	350
isophorone			9	0	690			11	0	350
2-nitrophenol			9	0	690			11	0	350
2,4-dimethylphenol			9	0	690			11	0	350
benzoic acid			9	0	2,792			11	0	1,700
bis(2-chloroethoxy)methane			9	0	690			11	0	350
2,4-dichlorophenol			9	0	690			11	0	350
1,2,4-trichlorobenzene			9	0	690			11	0	350
naphthalene			9	0	690			11	0	350
4-chloroaniline			9	0	690			11	0	350
hexachlorobutadiene			9	0	690			11	0	350
4-chloro-3-methylphenol			9	0	690			11	0	350
2-methylnaphthalene			9	0	690			11	0	350
hexachlorocyclopentadiene			9	0	690			11	0	350
2,4,6-trichlorophenol			9	0	690			11	0	350

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 4 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
2,4,5-trichlorophenol			9	0	3,300			11	0	1,700
2-chloronaphthalene			9	0	690			11	0	350
2-nitroaniline			9	0	3,300			11	0	1,700
dimethylphthalate			9	0	690			11	0	350
acenaphthylene			9	0	690			11	0	350
2,6-dinitrotoluene			9	0	690			11	0	350
3-nitroaniline			9	0	3,300			11	0	1,700
acenaphthene			9	0	690			11	0	350
2,4-dinitrophenol			9	0	3,300			11	0	1,700
4-nitrophenol			9	0	3,300			11	0	1,700
dibenzofuran			9	0	690			11	0	350
2,4-dinitrotoluene			9	0	690			11	0	350
diethylphthalate			9	0	690			11	0	350
4-chlorophenyl-phenylether			9	0	690			11	0	350
fluorene			9	0	690			11	0	350
4-nitroaniline			9	0	3,300			11	0	1,700
4,6-dinitro-2-methylphenol			9	0	3,300			11	0	1,700
N-nitrosodiphenylamine (1)			9	0	690			11	0	350
4-bromophenyl-phenylether			9	0	690			11	0	350
hexachlorobenzene			9	0	690			11	0	350

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 5 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
pentachlorophenol			9	0	3,300			11	0	1,700
phenanthrene			9	0	690			11	0	350
anthracene			9	0	690			11	0	350
di-n-butylphthalate			9	0	690			11	0	350
fluoranthene			9	0	690			11	0	350
pyrene			9	0	690			11	0	350
butylbenzylphthalate			9	0	690			11	0	350
3,3'-dichlorobenzidine			9	0	690			11	0	710
benzo(a)anthracene			9	0	690			11	0	350
chrysene			9	0	690			11	0	350
bis(2)-ethylhexyl)phthalate			9	0	690			11	0	350
di-n-octylphthalate			9	0	690			11	0	350
benzo(b)fluoranthene			9	0	690			11	0	350
benzo(k)fluoranthene			9	0	690			11	0	350
benzo(a)pyrene			9	0	690			11	0	350
indeno(1,2,3-cd)pyrene			9	0	690			11	0	350
dibenz(a,h)anthracene			9	0	690			11	0	350
benzo(g,h,i)perylene			9	0	690			11	0	350
Pesticides										
alpha-BHC			9	0	17			11	0	17
beta-BHC			9	0	17			11	0	17

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 6 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
delta-BHC			9	1	14			11	0	17
gamma-BHC (indane)			9	0	17			11	0	17
heptachlor			9	0	17			11	0	17
aldrin			9	0	17			11	0	17
heptachlor epoxide			9	0	17			11	0	17
endosulfan I			9	0	17			11	0	17
dieldrin			9	0	33			11	0	34
4,4'-DDE			9	0	33			11	0	34
endrin			9	0	33			11	0	34
endosulfan II			9	0	33			11	0	34
4,4'-DDD			9	0	33			11	0	34
Aniline			9	0	33			11	0	34
endosulfan sulfate			9	0	33			11	0	34
4,4'-DDT			9	0	33			11	0	34
methoxychlor			9	0	170			11	0	170
endrin ketone			9	0	33			11	0	34
alpha-chlordane			9	0	170			11	0	170
gamma-chlordane			9	1	160			11	0	170
toxaphene			9	0	330			11	0	340
aroclor-1016			9	0	170			11	0	170
aroclor-1221			9	0	170			11	0	170

Table IV-9. TCL Parameter UTL's for Background Soils (μg/kg). (Sheet 7 of 7)

Parameter	Operable Unit Specific Background									
	>2 ft deep[1]					>2 ft deep[1]				
	$\bar{X}$	s	n	d	UTL	$\bar{X}$	s	n	d	UTL
aroclor-1232			9	0	170			11	0	170
aroclor-1242			9	0	170			11	0	170
aroclor-1248			9	0	170			11	0	170
aroclor-1254			9	0	330			11	0	340
aroclor-1260			9	0	330			11	0	340

X = Mean
s = standard deviation
n = number of samples
d = number of detects
UTL = upper 95 percentile tolerance limit
[1]Does not include saturated soils.
NA = Not analyzed for.

This page left intentionally blank.

Table 3-1. Maximum Concentrations for Detected Compounds, Compared to UTLs for Surface Soils (0 to 2 feet) from Phase I and II Data. (Sheet 1 of 3)

Parameter	Surface Soil UTL	Max Value 1100-1	Max Value 1100-2	Max Value 1100-3	Max Value 1100-4	Max Value 1100-6	Max Value HRL	Max Value EP
INORGANIC COMPOUNDS (mg/kg)								
Aluminum	9708.79	7130	8300	**9770**	7320	8680	**15800**[b]	5810
Antimony	3.70	ND	ND	ND	ND	ND	**15.6**[b]	ND
Arsenic	3.99	3.2	2.3	3.4	2.6	2.7	3.6	2.6
Barium	120.10	80.8	91.5	106	80.9	99.2	**1320**	72.3
Beryllium	0.74	ND	0.51	0.44	0.25	0.4	**1.3**	0.26
Cadmium	0.70	ND	ND	ND	ND	ND	**2**	ND
Calcium	5128.25	**8690**	**6480**	**6810**	**9710**	4180	**86700**	3030
Chromium	12.94	10.6	**16.8**	14	11.3	10.9	**17.1**	7.7
Cobalt	17.74	13.2	13.9	14.1	11.4	12.2	15.9[b]	10.3
Copper	18.11	**37.9**	**24.4**	**22.8**	14.4	16.2	**58.6**	15.2
Iron	31110.42	21100	26600	25500	23300	23500	29800	18900
Lead	12.64	**266**	**94.6**	**26.4**	5	**22.1**	**482**	54.2
Magnesium	6523.59	6430	5210	6170	4650	4840	**25000**	4250
Manganese	552.27	484	385	436	330	383	423	354
Mercury	0.10	**0.22**	ND	ND	ND	ND	**1.3**	ND
Nickel	19.00	**20.9**	15	14.9	9.8	12.9	**174**	12.5
Potassium	1909.71	850	**2060**	1730	1210	**1950**	**2230**	1140
Selenium	0.39	ND	ND	ND	ND	ND	**0.97**[b]	ND
Silver	2.44	ND	ND	ND	ND	ND	**4.5**	ND
Sodium	241.52	**479**	**374**	**495**	**413**	143	**5140**[b]	216
Thallium	0.39	ND	**0.48**	**.40**	ND	ND	**.42**	ND
Vanadium	83.93	32.5	73.4	70.2	61.8	60.8	**87.3**	44.4
Zinc	62.20	**92**	56.6	59	45.9	**111**	**408**	**67.5**
Cyanide	0.52	ND	ND	ND	ND	ND	**0.56**	ND
VOLATILE ORGANIC COMPOUNDS (µg/kg)								
1,1,1-trichloroethane	5	ND	2	ND	ND	**35**	ND	ND
1,1-dichloroethene	5	ND	**5**	ND	ND	ND	ND	ND
2-butanone	11	ND	**10**[a]	**17**[a]	ND	**69**[a]	**35**[a,b]	ND
2-hexanone	11	ND	ND	ND	ND	**53**	ND	ND
Acetone	43	ND	**19**[a]	**92**[a]	**6**[a]	**190**[a]	ND	ND
Chlorobenzene	5	ND	**6**	ND	ND	ND	ND	ND
Methylene chloride	5	ND	**42**[a]	**120**[a]	ND	**20**[a]	**43**[a]	**4**[a]
Tetrachloroethene	5	ND	**35**	ND	ND	ND	**5**	ND
Toluene	5	ND	**11**[a]	**6**[a]	ND	**8**[a]	**16**[a]	ND
Trichloroethene	5	ND	**6**	ND	ND	ND	ND	ND
Xylene	5	ND	**6**	ND	ND	ND	ND	ND

Table 3-1. Maximum Concentrations for Detected Compounds, Compared to UTLs for Surface Soils (0 to 2 feet) from Phase I and II Data. (Sheet 2 of 3)

Parameter	Surface Soil UTL	Max Value 1100-1	Max Value 1100-2	Max Value 1100-3	Max Value 1100-4	Max Value 1100-6	Max Value HRL	Max Value EP
SEMI-VOLATILE ORGANIC COMPOUNDS (µg/kg)								
1,2,4-trichlorobenzene	690	ND	120	ND	ND	83	ND	ND
1,3-dichlorobenzene	690	ND	120	ND	ND	ND	ND	ND
1,4-dichlorobenzene	690	ND	120	ND	ND	86	ND	ND
2-chlorophenol	690	ND	230	ND	ND	170	ND	ND
2-methylnaphthalene	690	ND	ND	ND	ND	ND	**7100**	ND
2,6-dinitrotoluene	690	ND	ND	ND	ND	ND	210[b]	ND
4-chloro-3-methylphenol	690	ND	190	ND	ND	95	ND	ND
4-nitrophenol	3300	ND	ND	ND	ND	ND	**3800**	ND
Acenaphthene	690	ND	110	ND	ND	77	ND	ND
Anthracene	690	ND	ND	ND	ND	ND	70[b]	ND
Benzoic acid	2790	ND	ND	ND	ND	ND	220[a]	ND
Benzo(a)anthracene	690	ND	ND	120	ND	ND	180	ND
Benzo(a)pyrene	690	ND	110	150	ND	ND	200	ND
Benzo(b)fluoranthene	690	150	79	180	ND	ND	250	ND
Benzo(g,h,i)perylene	690	ND	330	230	ND	ND	150	ND
Benzo(k)fluoranthene	690	ND	120	160	ND	ND	190	ND
Bis(2-ethylhexyl)phthalate	690	390[a]	290[a]	940[a]	ND	**2.5E+07**	ND	ND
Butylbenzylphthalate	690	ND	ND	ND	ND	ND	99[a]	ND
Chrysene	690	100	ND	170	ND	ND	240	ND
Dibenzofuran	690	ND	ND	ND	ND	ND	130	ND
Dibenz(a,h)anthracene	690	ND	300	110	ND	ND	ND	ND
Di-n-butyl phthalate	690	ND	ND	ND	ND	ND	65[b]	ND
Di-n-octyl phthalate	690	ND	67[a]	ND	ND	**46000**	ND	ND
Fluoranthene	690	110	ND	220	ND	ND	180	ND
Indeno(1,2,3-cd)pyrene	690	ND	300	230	ND	ND	170	ND
Naphthalene	690	ND	ND	ND	ND	ND	1100	ND
N-nitroso-di-n-propylamine	690	ND	110	ND	ND	78	ND	ND
Pentachlorophenol	3300	ND	ND	99	ND	ND	980[b]	ND
Phenanthrene	690	ND	ND	130	ND	ND	380[a]	ND
Phenol	38100	ND	94	ND	ND	ND	ND	ND
Pyrene	690	97	120	250	ND	94	220	ND

Table 3-1. Maximum Concentrations for Detected Compounds, Compared to UTLs for Surface Soils (0 to 2 feet) from Phase I and II Data. (Sheet 3 of 3)

Parameter	Surface Soil UTL	Max Value 1100-1	Max Value 1100-2	Max Value 1100-3	Max Value 1100-4	Max Value 1100-6	Max Value HRL	Max Value EP
PESTICIDES/PCBs (µg/kg)								
4,4"-DDE	33	6.8	**42**	ND	ND	**170**	**1200**	ND
4,4'-DDD	33	ND	3.6	ND	ND	ND	**260**	ND
4,4'-DDT	33	ND	**57**	ND	ND	ND	**520**[b]	ND
Aldrin	17	ND	9.6[a]	1.1[a]	ND	9.6[a]	11[b]	ND
Alpha-chlordane	170	6.5	ND	ND	ND	**1000**	**770**[b]	**1100**[b]
Total PCBs	1510	290	300	150	ND	ND	**100550**	**42000**
Aroclor 1248	170	ND	ND	ND	ND	ND	**100000**[b]	ND
Aroclor 1260	330	290	300	150	ND	ND	260	**42000**[b]
Aroclor-1254	330	ND	ND	ND	ND	ND	290	ND
Beta-BHC	17	ND	ND	ND	ND	ND	**94**[b]	ND
Delta-BHC	14	ND	ND	ND	ND	13	ND	ND
Dieldrin	33	ND	1.3	ND	ND	2.3	**1200**[b]	ND
Endosulfan II	33	ND	ND	ND	ND	ND	**110**[b]	160
Endosulfan sulfate	33	ND	ND	ND	ND	ND	19	ND
Endrin	33	ND	ND	ND	ND	ND	**280**[b]	**39**
Endrin ketone	33	ND	2	ND	ND	1.3	**140**[b]	ND
Gamma-BHC(Lindane)	17	ND	ND	ND	ND	0.77	1.9	ND
Gamma-chlordane	158	6.2	ND	ND	ND	**860**	82	**1700**[b]
Heptachlor	17	ND	1.2	ND	ND	**65**	ND	29
Methoxychlor	170	ND	ND	ND	ND	ND	140[b]	ND

ND - Contaminant not detected
UTL - Upper tolerance limit
[a]Concentration less than detection limit after blank-adjustment
[b]Phase II data

This page left intentionally blank.

Table IV-11. Maximum Concentrations for Detected Compounds Compared to UTL's for Subsurface Soils (> 2 feet) from Phase I and Phase II Data. (Sheet 1 of 2)

Parameter	Subsurface Soil UTL	Max Value 1100-1	Max Value 1100-2	Max Value 1100-3	Max Value 1100-4	Max Value 1100-6	Max Value HRL	Max Value EP
INORGANICS (mg/kg)								
Aluminum	6236	5860	**7470**	**7400**	**6680**	NS	**17800**[b]	NS
Antimony	3.1	ND	3	ND	ND	NS	**15.6**[b]	NS
Arsenic	2.92	**3.2**	1.8	1.8	**5.8**	NS	**6.6**	NS
Barium	236	85.9	96.6	85.9	98.7	NS	**511**[b]	NS
Beryllium	0.27	ND	ND	ND	**0.93**	NS	**1.1**[b]	NS
Cadmium	0.36	ND	ND	ND	ND	NS	**2.4**[b]	NS
Calcium	7830	6240	**13000**	**9080**	**10600**	NS	**44800**[b]	NS
Chromium	47.3	14.6	10.3	13.6	13.2	NS	**1,250**	NS
Cobalt	16.8	11.8	15.3	**17.8**	16.5	NS	**42.5**	NS
Copper	19.5	**25**	**23.6**	**31.7**	**19.8**	NS	**1280**[b]	NS
Cyanide	0.51	ND	ND	ND	ND	NS	**0.56**	NS
Iron	29400	25800	27100	**31700**	26700	NS	**35200**	NS
Lead	5	**191**	**45.9**	4.7	**5.7**	NS	**854**[b]	NS
Magnesium	4680	3860	4620	**5290**	4630	NS	**7640**[b]	NS
Manganese	355	249	**366**	381	329	NS	**501**[b]	NS
Mercury	0.1	**0.39**	ND	ND	ND	NS	**0.44**	NS
Nickel	26	9.5	13.8	11.3	10.7	NS	**557**	NS
Potassium	966	**4880**	**1200**	878	**1030**	NS	**3820**[b]	NS
Selenium	0.41	ND	ND	ND	ND	NS	0.36	NS
Silver	0.54	ND	ND	ND	2	NS	7.7	NS
Sodium	419	**808**	**458**	**999**	**726**	NS	**2360**[b]	NS
Thallium	0.41	ND	ND	ND	**0.48**	NS	**0.46**	NS
Vanadium	115	**118**	80.2	103	82.4	NS	101	NS
Zinc	50.4	**100**	**54.9**	**60**	**63.8**	NS	**3,160**[b]	NS
VOLATILE ORGANIC COMPOUNDS (µg/kg)								
2-butanone	11	9[a]	8[a]	**11**[a]	ND	NS	**23**[a]	NS
Acetone	22	**26**[a]	**28**[a]	**29**[a]	9[a]	NS	**200**	NS
Benzene	5	ND	ND	ND	ND	NS	0.3[b]	NS
Ethylbenzene	5	ND	2	ND	ND	NS	ND	NS
Methylene Chloride	5	ND	**61**[a]	**16**[a]	ND	NS	**5**[a]	NS
Tetrachloroethene	5	ND	**15**[b]	ND	ND	NS	4[b]	NS
Toluene	5	ND	3[a]	ND	ND	NS	ND	NS
SEMI-VOLATILE ORGANIC COMPOUNDS (µg/kg)								
1,2,4-trichlorobenzene	350	ND	ND	ND	ND	NS	230[b]	NS
1,4-dichlorobenzene	350	ND	ND	ND	ND	NS	170	NS
2-chlorophenol	350	ND	ND	ND	ND	NS	240[b]	NS
2,4-dinitrotoluene	350	ND	ND	ND	ND	NS	92	NS
4-choro-3-methyphenol	350	ND	ND	ND	ND	NS	290	NS
4-nitrophenol	1700	ND	ND	ND	ND	NS	310	NS
Acenaphthene	350	ND	ND	ND	ND	NS	320[b]	NS
Benzoic Acid	1700	ND	ND	ND	ND	NS	160[a,b]	NS
Benzo(b)fluoranthene	350	74	ND	ND	ND	NS	ND	NS
Bis(2-ethylhexyl) phthalate	350	ND	**3600**[a]	**950**[a]	ND	NS	**1,000**[a]	NS
Di-n-butylphthalate	350	ND	37	ND	ND	NS	ND	NS
Di-n-octylphthalate	350	ND	ND	ND	ND	NS	270[a,b]	NS
Fluoranthene	350	110	ND	ND	ND	NS	ND	NS
N-nitro-di-n-propylamine	350	ND	ND	ND	ND	NS	170	NS
Pentachlorophenol	1700	ND	ND	ND	ND	NS	280	NS
Phenol	350	ND	ND	ND	ND	NS	330[b]	NS
Pyrene	350	84	290	ND	ND	NS	270[b]	NS

Table IV-11. Maximum Concentrations for Detected Compounds Compared to UTL's for Subsurface Soils (> 2 feet) from Phase I and Phase II Data. (Sheet 2 of 2)

Parameter	Subsurface Soil UTL	Max Value 1100-1	Max Value 1100-2	Max Value 1100-3	Max Value 1100-4	Max Value 1100-6	Max Value HRL	Max Value EP
PESTICIDES (µg/kg)								
Aldrin	17	ND	16[a]	ND	ND	NS	5.5[a,b]	NS
Alpha-chlordane	170	1.3	ND	ND	ND	NS	13[b]	NS
4,4'-DDE	34	ND	**39**	ND	ND	NS	14	NS
4,4'-DDT	34	ND	**121**	ND	ND	NS	ND	NS
Beta BHC	17	ND	ND	ND	ND	NS	1.2[b]	NS
Dieldrin	34	ND	ND	ND	ND	NS	**90**[b]	NS
Endrin	34	ND	ND	ND	ND	NS	**120**[b]	NS
Endrin ketone	34	ND	22	ND	ND	NS	ND	NS
Heptachlor	17	ND	ND	0.58	ND	NS	ND	NS
Total PCB's	1530	ND	160	ND	ND	NS	**2640**	NS
Aroclor 1248	170	ND	ND	ND	ND	NS	**640**	NS
Aroclor 1254	340	ND	ND	ND	ND	NS	**2,000**[b]	NS
Aroclor 1260	340	ND	160	ND	ND	NS	ND	NS

Notes:
ND: contaminant not detected
UTL: upper tolerance limit
NS: no subsurface samples collected for analysis
[a]Concentration less than detection limit after blank - adjustment
[b]Phase 2 data

DATA REPORTING QUALIFIERS

The following is a summary of data reporting qualifiers and abbreviations used in the tables for this appendix.

B - Organic Samples: Indicates compound was found in the associated blank as well as in the sample.

Inorganic Samples: Indicates value is greater than the instrument detection limit and below the contract required detection limit.

J - Indicates an estimated value.

U - Indicates compound was analyzed for but not detected at the given detection limit. Values associated with a U qualifier are one-half the SQL.

R - Data has been rejected during the validation process.

ABBREVIATIONS

-- Data result not used (see groundwater discussion Section 2, Appendix E)

UCL - Upper confidence limit of 95 used in the statistical calculations.

SDG - Sample delivery group.

UNK - Location is unknown.

NA - Analysis not performed, not available, or not used in the risk assessment.

NR - Not requested for analysis.

*Chlordane - The concentrations reported for alpha and gamma chlordane were summed.

SQL - Sample quantitation limit.

S - Surface sample.

WHC - Westinghouse Hanford Company.

< - Indicated the radioactivity is less than the given count.

Q - Data qualifier indicating acceptability for use in risk assessment; (a blank indicates no associated qualifier).

1.1 UN-1100-6 SUBUNIT (DISCOLORED SOIL SITE)

Bis(2-ethylhexyl)phthalate (BEHP) and Chlordane

Alpha and gamma chlordane were summed for statistical calculations. Data for BEHP and chlordane were treated in the same way since their distributions on the site are similar. BEHP and chlordane were detected in samples A6150S to A6155S and were greater than any other detections. Because these samples are all in close proximity to each other, only data from these samples were used for statistical calculations. Data used in the calculations are provided in table E-2. The use of these data provides a conservatively biased estimate of the 95 percent UCL because low values or nondefects are not used.

1.2 EPHEMERAL POOL

Chlordane and PCB's

All data for these contaminants, collected from this site, were used in the calculations. The data are summarized in table E-3.

1.3 HRL

Arsenic and Beryllium

These contaminants are evenly distributed on the site. All data were included that were taken from the surface to a depth of 15 feet.

Chromium

In borehole HRL-4, chromium was found to be at a significantly higher concentration than any of the other samples on the site. In order to estimate the concentrations over the 15-foot soil column, data taken from all boreholes and trenches down to 15 feet were used in calculations. Data from auger holes and surface samples not associated with boreholes were not used to calculate the 95 percent UCL. These data provide a conservatively biased estimate of the 95 percent UCL for evaluation of chromium.

PCB's

Elevated levels of PCB were mostly found in close proximity to HRL-4, therefore the 95 percent UCL calculations used data from samples taken from this vicinity. Data used were from AH203, Borehole HRL-4 (0-2.8 feet), PCB-1 to PCB-4 and PCB-1A to PCB-4A.

The data for the HRL used to calculate the 95 percent UCL are presented in table E-4.

2.0 CALCULATION OF 95 PERCENT UCL FOR GROUNDWATER CONTAMINANTS

The 95 percent UCL's for contaminants in the groundwater in the vicinity of the HRL were calculated as described above. Two nonradioactive contaminants are evaluated. These contaminants are trichloroethene and nitrate. In addition, gross alpha and gross beta are evaluated because they have been detected at elevated concentrations in some sampling rounds as discussed in Section 5. For radioactive contaminants, actual net counts were used in the tables.

2.1 NONRADIOACTIVE CONTAMINANTS

Trichloroethene (TCE)

Data from MW-12 to MW-15 were used for statistics, because concentrations of TCE are consistently detected over MCL (5 mg/L) at these wells. The use of these data provide a conservatively biased 95 percent UCL of groundwater quality within the contaminant plume.

Nitrate (as Nitrogen)

Statistics are performed on data from MW-10 to MW-15 and MW-20 because nitrate was detected above MCL (10 mg/L) at these wells. Other data for nitrate were not used to calculate the 95 percent UCL. As indicated above, this provides a conservatively biased estimate of the groundwater quality within the contaminant plume.

The 95 percent UCL's are summarized in table E-5. The data used to calculate the 95 percent UCL's are presented in table E-6.

2.2 RADIOACTIVE CONTAMINANTS

Gross alpha and gross beta contamination have also been detected in the groundwater in the vicinity of HRL. As discussed in Chapter 5 of the risk assessment, most of the beta activity appears to be associated with Technetium-99. The 95 percent UCL's for gross alpha and gross beta activity are summarized in table E-5. Data from wells located within the contaminant plume were used to estimate conservatively biased 95 percent UCL's. In general, gross alpha activity exceeded 5 pCi/L or gross beta activity exceeded 50 pCi/L at the wells used for the calculation of the 95 percent UCL's. These activity levels are not MCL's, but are concentration limits with which the assumption of compliance with radionuclide MCL's may be assumed without further analysis.

The data used to calculate the 95 percent UCL's are presented in table E-7. The wells used to calculate the 95 percent UCL's for gross alpha are MW-10 to MW-15. The wells used to calculate the 95 percent UCL's for gross beta are MW-10 to MW-15 and MW-20.

3.0 UPPER TOLERANCE LIMIT

The tolerance interval is a statistical interval that contains at least a specified proportion, p, of the population with a specified degree of confidence, $100(1-\alpha)$ percent (Hahn and Meeker, 1991). Thus, the tolerance interval provides an estimate of the limits which define a proportion of the population, in contrast to the confidence interval which provides an estimate of a population parameter (*e.g.*, mean or variance). As the sample size, n, approaches infinity, the width of the tolerance interval approaches a finite range determined by the tolerance limits. In contrast, the width of a confidence interval approaches zero as n increases (Hines and Montgomery, 1980).

The UTL is an upper bound on the tolerance interval and, therefore, provides an estimate of the maximum expected value for the specified proportion of the population. This UTL is calculated using the equation:

$$UTL = X + Ks$$

where UTL is the upper tolerance limit, X is the sample mean, K is the tolerance factor, and s is the sample standard deviation. Values for K are found in appropriate tables in Hahn and Meeker, 1991, and are based on specified values for the population proportion (p), confidence ($1-\alpha$), and the number of samples (n) used to calculate the mean and standard deviation.

For this risk assessment, the UTL was calculated for surface soils (1 to 2 feet) and subsurface soils (> 2 feet) to provide a representation of analyte concentrations that could be expected in samples that have been unaffected by activities associated with the 1100-EM-1 Operable Unit (background). Comparison of analyte concentrations in samples collected from within the operable unit with the appropriate analyte UTL determined which analytes are greater than background and must be considered contaminants.

The UTL's were calculated to contain 95 percent of the population (p) with a 95 percent degree of confidence (α=0.05). Tables IV-8 and IV-9 contain the sample mean (X), sample standard deviation (s), number of background samples analyzed (n), the number of background samples in which the analyte is detected (d), and the UTL for the target analyte list (TAL) and target compound list (TCL) analytes, respectively. Background sample data used to generate the statistical values are contained in appendix I of the 1100-EM-1 Phase I RI (DOE-RL, 1990). The samples used to calculate UTL's for surface soils are: AH217S, AH218S, AH222S, AH224S, AH225S, A0201S, A0101, A0301S. The samples used to calculate UTL's for subsurface soils are A0203S, A0204S, A0206S, A0208S, A0209S, A0210S, A0302, A0306, A0104, A0105, A0109S. For those analytes not detected in any sample, the highest sample quantitation limit (SQL) was used as the UTL. If an analyte was detected in at least one sample, the mean and standard deviation were calculated; one-half of the SQL is used as a surrogate sample value for those samples where the analyte was reported as nondetectable in this case. This is consistent with DOE-RL, 1992.

Tables IV-10 and IV-11 provide a comparison between the UTL and the maximum concentration for contaminants detected in surface and subsurface soil samples, respectively,

from the various subunits. These tables incorporate data that was collected during the Phase I and Phase II Operable Unit RI.

4.0 REFERENCES

DOE/RL, 1990.

DOE/RL, 1992.

Hahn and Meeker, 1991

Hines, W.W., and D.C. Montgomery, 1980, *Probability and Statistics in Engineering and Management Science*, 2nd ed., John Wiley & Sons, Inc., New York, New York.

THIS PAGE INTENTIONALLY LEFT BLANK